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MINNESOTA MEDICINE

Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society.

VOL. XII DECEMBER, 1929 No. 12

THE RECOGNITION AND TREATMENT OF RENAL LITHIASIS*

ARTHUR L. CHUTE, M.D.
Boston

IT is often difficult to say what it was that determined the formation of a renal stone in a given individual. An increase of some normal constituent of the urine, either an "actual" increase due to the presence of an abnormally large amount of the material, or a "relative" increase due to a decreased amount of fluid, is probably an important factor in this process of stone formation. Little anomalies of the kidney such as a dilated lower calyx or a kidney pelvis that does not quite empty itself probably cause a degree of urinary stasis that favors the precipitation of the mineral constituents that form stones. The presence of some albuminous material is said to favor this precipitation; by some it is said to be essential for its occurrence. Renal infections, especially those infections with the urea-splitting organisms, that produce an alkaline urine, are apparently a determining factor in many cases. Whatever our theory as to the formation of renal stones, we are faced by the fact that the condition is a common one, and one that is responsible for a great deal of suffering as well as a very great destruction of kidney tissue.

Their destructive power is so great that I believe it is fair to say any stone that remains indefinitely in a kidney and has the increase in size that these stones ordinarily have, will lead in the end to that kidney's total destruction. Not only this, but the danger of a renal stone that has passed into a ureter, and is, strictly speaking, a ureteral stone, is to be computed solely in terms of the risk to which it subjects its kidney. Furthermore, not only the danger but the pain a ureteral stone causes is due not to the trauma that it inflicts on the ureter, but to the degree that it distends the kidney pelvis. I shall have occasion to refer to this point later. A stone that had its origin in a kidney does not cease to menace that kidney until it has reached the bladder.

Within a short time I have had impressed upon me, through the death of a patient whom I had seen over a series of years, the slow but inexorable progress, even to a fatal termination, that urinary lithiasis may make unless recognized early and successfully treated. The patient in point, a woman of 58, whom I first saw in 1920, came with the story that for the previous five or six months she had been having pain in her right side. Examination showed a shadow of moderate size in the pelvis of her right kidney, which also showed a moderate degree of infection. Her left kidney contained the shadow of a large stone and was found to be destroyed functionally. It excreted nothing but moulds of pus. I removed by pyelolithotomy the stone in her right kidney pelvis and the patient made a good surgical convalescence. There was no history of pain at any time in her left kidney, the destroyed side; this I did not touch.

About three years after her pyelolithotomy this patient had a recurrence of symptoms in her right side. Even then it was found she had a phenolsulphonephthalein output of about 45 per cent. Again I operated upon her and removed two small stones. This time a nephrolithotomy was done. The patient had a stormy surgical convalescence, but from 1923 to the early part of 1928 was fairly well with the exception of occasional upsets, presumably due to a moderate degree of renal insufficiency. By the latter part of 1928 she was nauseated, markedly anemic, had a moderate amount of edema, and was found to have practically no output of phenolsulphone-

*Presented before the annual meeting of the Minnesota State Medical Association, St. Paul, May 14, 1929.

phthalein. She failed and died a short time later of renal insufficiency.

This patient's history illustrates strikingly several important things regarding urinary stones: (1) the lack of subjective symptoms that these patients with renal stones sometimes show, as illustrated by the condition in her left kidney, of which she had apparently never had any intimation; (2), the relentlessness of the process when not interfered with, as illustrated by the total destruction of the same kidney; (3) an unfortunate tendency that renal stones have to be bilateral; and finally our inablity to stop the destructive process after it gets beyond a certain point as illustrated by the fact that though at the second time I removed stones from her right kidney she had a very fair renal function and though from then, until the time of her death, this patient had no symptoms of any reformation of stones, yet the destructive process continued relentlessly until she had practically no renal function.

The most important point to my mind in this woman's history is the fact that her left kidney was destroyed when I first saw her, as proven by the radiographs that showed a large stone and by cystoscopy that showed the kidney had no urinary excretion; that it simply pushed out from time to time moulds of pus, and yet this patient said she had never had any symptoms referred to that side. I do not remember whether or not I went over the question of the process having been absolutely symptomless in great detail but I believe we may accept as a fact that it had never given any symptoms that had suggested the possibility of renal stone to either this patient or her doctor.

It is not a very uncommon thing to find a patient who has had a kidney practically destroyed by a stone and yet has had no symptoms that were sufficiently clearly marked to give a clue as to what was taking place. This I believe is our most serious difficulty in preventing the very great destruction that urinary stones, especially renal stones, do, since often they are not recognized until irreparable injury has been done.

This is not due to the fact that these stones do not actually produce symptoms but is due to the fact that they often do not produce symptoms which the man in general practice, to whom these patients almost invariably appeal first, has been accustomed to recognize as symptoms of renal stone. In the case of the patient I have cited it may perfectly well have been that all the discomfort she had suffered had been backache, a symptom common with women, together perhaps with a turbid urine, also easily overlooked in women.

For many generations before the introduction of the *x*-ray, two symptoms were associated with renal stone, excruciating pain in a loin or back and hematuria; and today many men find it hard to accept less striking symptoms as an indication of renal stone. It is true that excruciating pain may be met in such renal stones as block the kidney outlet and with this pain there is often associated more or less bleeding. In my experience, however, the excruciating pain commonly called colic is more often seen in patients in whom a stone has left a kidney and has started down a ureter, and where from time to time, and for no reason that we can clearly see, the stone is held by spasm of the irritated ureter. This spasm of the ureter leads to an acute dilatation of that section of the ureter above the stone and the corresponding kidney pelvis and to the occurrence of an acute hydronephrosis that is most painful. I have on a number of occasions been able to work a ureter catheter past a stone held by spasm in a ureter and draw off the urine that was distending the kidney pelvis; this has stopped literally within a minute or two a very acute pain that had been imperfectly controlled even by large doses of morphia. In one such instance, seen some years ago, after working a ureter catheter by a stone impacted in a ureter I withdrew from the kidney pelvis between 50 and 60 c.c. of urine with immediate relief to the patient's pain. The next day when I did a pyelogram, this same kidney pelvis would hold only between 6 and 8 c.c. of fluid. It is not surprising that such a marked overdistension should cause great pain.

As to the presence of hematuria, in a series of cases that I went over a few years ago, bleeding that was evident to the eye had been present in but 30 per cent of the cases; microscopic blood had been found in 41 per cent, though doubtless it was present at some time in all or practically all the cases. However valuable microscopic blood may be in confirming a diagnosis, once it has been suspected, it has but little suggestive value. Unfortunately the stones held

firmly in a kidney, the large ones that cause atrophy of kidney tissue by pressure as they increase in size and predispose to suppuration, the ones that are especially destructive, do not cause the marked symptoms that are brought about by the smaller stones that block the kidney outlet and produce acute dilatation of the kidney pelvis. These large stones may produce only a moderate degree of pain, usually a backache, and this not necessarily a constant pain but one that may recur at periods over a very long time. In the case of two patients from whom I removed wholly disorganized kidneys for calculous pyonephrosis, the symptom of backache had recurred over periods of twenty and thirty-five years respectively before these patients had made a real determined effort to find the cause of the discomfort and be relieved of it. A man, from who I removed a kidney which had been reduced to a mere shell by a calculous hydronephrosis, had only the history of a short attack of pain in the same loin some fourteen years before, then freedom from all symptoms up until about two weeks before he had come to me, when he had a slight hematuria.

Another man from whom I removed a renal stone some years ago with relief of his symptoms, had suffered from pain in the upper abdomen, treated as dyspepsia over a period of twenty years; it was not until he applied for life insurance and it was found that his urine contained pus that the cause of his trouble was recognized and remedied.

Last fall a somewhat similar case came under my care. This was a man of fifty, who had suffered from "indigestion" for four years and in whose urine pus had been found when he had applied for life insurance. The pus was found to depend upon a stone in his left kidney, which was removed. At last report he was free from his indigestion. These are not excessively rare examples of the confusing subjective symptoms that patients with renal stone may present; in fact a predominance of digestive symptoms occurred in 15 per cent of a small series of cases of renal stone that I went over some years ago. In a few instances of stone in a kidney the subjective symptoms, if there are any, will be so vague as not to suggest the condition and the only objective sign may be a turbid urine.

In spite of the more unusual cases, however, pain in varying degree is the most common sign of a kidney stone. This pain may be in the back, loin, or abdomen, and vary from the excruciating and characteristic pain of kidney colic to a slight grumbling discomfort. A history of blood in the urine will be present in about a third of the cases. Bleeding is much more suggestive if it appears regularly as a result of exercise.

In a condition that is so common and so destructive and yet in which the signs may be so vague, we are most fortunate in having so accurate a diagnostic means as we have in radiography. Much as the surgeon or the general practitioner may dislike to make the confession, we shall have to admit that we are wholly dependent upon the help of the radiographer for early and accurate diagnosis in most cases of renal stone. We may suspect the condition but that is as far as we can go unaided in a very large proportion of cases. A good radiograph, however, in a patient who is not too fat and who has been properly prepared, should show a shadow in all cases of renal stone with the exception of a few small uric acid stones. Furthermore, as a good renal *x*-ray will show the outline of the kidney and its relation to the shadow of the stone, it will give a pretty good idea of the position that the stone occupies in the kidney.

We are so dependent upon the *x*-ray in our study of these cases that I shall consider this subject somewhat in detail. In order that we may not be led to draw false conclusions from radiography of the urinary tract, it is essential that certain precautions be observed in taking the plates. It is a very common thing when one is seeking the cause of some rather obscure abdominal pain to make use of a barium meal or a barium enema. If this investigation has been negative, the patient may then be radiographed as to the question of urinary stone. Under these circumstances bits of barium left in the intestine may so overlie the stone-bearing area of a kidney as to raise a question as to the nature of a given shadow. Therefore, it is wise, if one foresees the necessity of radiographs of the genito-urinary tract, to have these taken before gastro-intestinal plates are taken, of if barium plates have already been taken that an interval be allowed to elapse before renal plates are made. Another important point in the technic of taking *x*-ray plates, when the question is that of urinary stone, is to take the whole tract. Very often a patient will have symptoms so sharply referred to one or the

other kidney that radiographs may be taken solely of that kidney region. I have in mind two instances where this led to a most unfortunate outcome since in both these patients the painful kidney was removed, only to find that later radiographs showed that the renal pain was undoubtedly due to stones that were caught low down in the ureter of the corresponding side and were producing the renal pain by urinary back pressure. In every instance in which one has the urinary tract radiographed because of a suspected urinary stone, one should be sure that the first set of films cover the whole region of the kidneys and ureters down to and including the bladder; otherwise, one will occasionally overlook a ureteral stone that is causing kidney pain, or a ureteral stone that accompanies a renal stone. In the first instance it is possible that a kidney may be sacrificed needlessly; in the second, the removal of the renal stone would probably be followed by a urinary sinus until such time as the ureteral stone had been recognized and removed. Further radiographs may often be confined with advantage to a limited area of the urinary tract but the first renal x-ray should include the whole stone-bearing area of the urinary tract; it should also show the kidney outline and its stone-bearing area free from overlying gas shadows, otherwise the film will have but little negative value.

Some of the radiographs taken in patients whom we suspect of harboring renal stones will show no shadows; others will show a shadow. In what cases shall we accept the negative evidence as final and in what cases not? It has been pretty definitely proved that small uric acid stones may not be brought out by a radiograph, especially if they are lying in a collection of urine. Therefore, if a patient's symptoms have impressed one strongly with the probability of stone, it is wise, in the case of a negative film, to pass a ureter catheter up to the kidney in question and if it contains a residual urine, to draw this off and then take radiographs. If after the residual has been withdrawn, the plates are still negative, one may put a small amount of pyelographic medium into the kidney pelvis and take further plates. These may show in the case of a calculus a pale shadow, framed in a denser one; if these tests are negative, we may safely assume that the kidney in question contains no stone of any size. However, it is always well in a very suggestive case to "save one's exceptions" so to speak, as it has been my experience that a small uric acid stone will from time to time be passed from a kidney in which I have been wholly unable to demonstrate its presence. Under these conditions, however, the stones have been so small that their presence has been more of academic interest than of surgical importance. So much for the value of the negative evidence that x-rays may show.

When we come to the cases in which radiographs show positive shadows, our diagnostic work is by no means finished. We have to differentiate carefully between the shadows that indicate renal stones and the shadows that indicate other more or less common conditions. When we are considering the question of renal stones we are concerned only with shadows that overlie the kidney outline, my reason for insisting that a satisfactory renal radiograph must show this outline. There are certain branched, coral-like shadows that we see overlying a kidney outline which can be nothing but renal stones and it is unnecessary to go further as regards the question of diagnosis. There are also shadows, much the shape of a peach stone, that are very suggestive.

A number, however, of the shadows that we find overlying the kidney outline are not associated with the urinary tract at all. The first of these shadows that we shall consider are those made up of things that are in the intestine. One sometimes sees little hardened masses of feces or bits of bone or other foreign matter taken with the food. I have already spoken of the confusion which masses of barium may make. A large mass may quite conceal the stone-bearing area of a kidney while a small mass may give a shadow suggestive of stone. The various things in the intestine will, however, usually be found to have changed position in two radiographs. If doubt still exists, a vigorous clearing out of the bowel will remedy the condition. Intestinal gas gives a dark shadow instead of a light shadow such as stone gives and will not cause any particular confusion in this respect. Intestinal gas is, however, particularly likely to be held over a sensitive kidney, such as one harboring a stone, and cause trouble by obscuring its stone-bearing area. As with intestinal contents, intestinal gas will be likely to change its position in two radiographs and should, of course, be largely done

away with by an adequate pre-*x*-ray bowel preparation.

The two sorts of shadows that are most difficult to differentiate from a stone in a kidney are the shadows of gallstone and those of calcified abdominal glands. Gallstones can cause confusion only on the right side. Calcified abdominal glands are common and may be found all over the abdomen. Along the lower course of the ureter, in the anatomical pelvis, there may be not only calcified glands, but calcified vessels and phleboliths to differentiate from ureteral stones.

The density of the shadows cast by gallstones varies with the amount of calcium the stones contain. They vary somewhat in shape but are almost invariably round. A few may have a density which would be compatible with that of a kidney stone. Most gallstones, however, that we are called upon to differentiate from renal stones are easily recognized. The larger ones are made up of a series of concentric rings, the smaller ones of faint shadowy rings, and the very small ones of an accumulation of very faint rings. Some gallstone shadows will be seen to be clearly to the outside of the kidney outline. The Graham test will sometimes make it clear that a doubtful shadow, overlying the kidney outline, is really in the gall-bladder. In most instances of gallstones the position and general appearance of the shadow are such as to make it evident that it is not a renal stone. In an occasional case it may be necessary to have recourse to pyelography to determine the position that such a shadow bears to the renal pelvis and calyces. I have stated before, and still believe, it is much more common to see a gall-bladder opened where the real lesion is a kidney, than to have a kidney explored where the lesion is a gallstone.

The shadow of a calcified abdominal gland, supposed to be the end-result of a tuberculous invasion, is the commonest condition that I know to simulate the appearance of a urinary stone. Both the mesenteric and retroperitoneal glands may be involved in this process. These patients suffer from more or less abdominal pain, though it is supposed that the glands that are painful are not the calcified ones which the *x*-ray shows, but others in which the inflammatory process has not gone on to calcification but is still more or less acute. These shadows often raise the question of a renal or ureteral stone. They will, however, have a mottled or granular appearance that is quite characteristic; furthermore, a mesenteric gland, being movable, will often change its position materially in two films. A calcified retroperitoneal gland is more fixed than a mesenteric gland but has the same mottling. In doubtful cases it may be necessary to make use of a radiographic catheter or a pyelogram or both to distinguish between these shadows of calcified glands and renal or ureteral stones. These maneuvers will, however, usually make the condition quite clear.

A ureteral stone is simply a renal stone that has been extruded from the kidney; this is nature's way of curing the condition, and is what I suppose actually takes place in a very large proportion of cases. As a rule, a stone that has gone down into a ureter exposes its kidney to less danger than if it had remained in the kidney itself, since it does not produce the atrophy by pressure upon the renal tissue that such a stone does if it remains in the kidney and increases in size. A stone in a ureter may still, however, be a very great menace to a kidney. I have referred to one instance in which a stone caught in a ureter led to the total destruction of its kidney through the hydronephrosis that it had produced. When in its passage to the bladder a ureteral stone irritates the ureter and causes it to contract violently about it, we have an attack of renal colic: the very excruciating pain that was formerly considered typical of renal stone. This is often accompanied by a little hematuria and sometimes by pain referred to the scrotum on the corresponding side, if the stone is low in the ureter. In the intervals between these attacks of colic a ureteral stone may give little or nothing in the way of subjective symptoms.

When a ureteral stone is situated anywhere but the lowest part of a ureter, its radiographic diagnosis is usually very easy, but even so there are few ureteral stones that I care to cut down upon without verifying my diagnosis by the passage of an *x*-ray catheter. Usually this will be enough to show that the shadow of the catheter is in close apposition to the stone shadow. If this is not convincing, an "offset" plate consisting of two exposures on a single film, the second taken after moving the lamp a short distance laterally, will usually settle the question. If in such a plate the stone shadow bears the same re-

lation to the catheter in both exposures we may be sure that it is in the same plane and consequently in the ureter. A more conclusive test may be made by filling a ureter with pyelographic medium and withdrawing the catheter. A ureteral stone will often hold the column of fluid above it very sharply and it may also show a distinctly widened ureter. These methods of differentiation will have to be used most frequently in the case of doubtful shadows in the lower part of a ureter; they will usually allow one to make a very accurate diagnosis.

As I have stated, it is my belief that every renal stone, if allowed to remain indefinitely, will lead eventually to the destruction of the kidney harboring it, either through the atrophy of kidney tissue caused by its increasing size or by the infection that comes sooner or later. I do not believe, however, that every renal stone should necessarily be removed the moment it is discovered. There are some very small stones that may cause pain and even bleeding and yet it may be unwise to attempt their removal. On two occasions at least I have attempted the removal of very small renal stones that were very elusive. In my search for these I so injured the kidneys that I had to remove them in both instances. I found the stones in the removed kidneys.

The location of the stone, too, makes a difference as to its ease of removal. It will be much easier to find and remove a small stone that is in a kidney pelvis than it will one that is in a lower calyx. I have seen within a week a patient in whose left kidney I found a very small shadow, 3-4 mm., 16 months ago, and who has returned from time to time for observation and radiography. In her last *x*-rays taken in April the stone had moved into the kidney pelvis, from the lower calyx. It has not increased in size so far as we can see nor has it caused infection nor a degree of pain that has been disabling. A stone of this type, I believe, it is fair to watch for a long time in the hope and with the reasonable expectation that it will be passed. When it comes to a renal stone that is more than 5-6 mm., roughly a quarter of an inch, in its smallest diameter, I believe we should advise its removal unless there is some definite contra-indication. Any renal stone is a foreign body and not only this but a foreign body that is bound to increase in size and the excretory work of the kidney makes its ultimate infection almost certain.

A stone in an infected kidney should be removed fairly promptly. In a few instances, however, I have advised against the removal of a totally destroyed stone-kidney in a patient whose kidney was draining well and who, I thought, was too poor a risk.

Some interesting problems come up in dealing with renal stones; thus it is especially important to remove a stone in a single kidney; though the danger of operation is greater than if the patient had a wider margin of safety in the way of renal tissue, yet this lessened amount of renal tissue makes imperative the conservation of as much of it as is possible and justifies one in taking an increased immediate risk to this end.

With bilateral renal stones, and the condition is not uncommon (occurring in 12 per cent in a series of stone cases that I looked up some time ago), all one's renal tissue is in jeopardy, just as is the case with a stone in a single kidney, and one must urge operation strongly though the indication is perhaps not quite so imperative as in the case of a stone in a single kidney. When stones are present in both kidneys, the better kidney should be operated upon first, if one can decide which one is the better; this the function test will usually tell us. In many of these bilateral cases I believe a bilateral renal infection was the cause of the stone. At any rate it is very common to find both kidneys infected at the time we first see these patients.

With a stone in a kidney and another in the ureter of the same side, either the removal of both should be done at the same time or one should remove the renal stone first, as its removal will tend to stop the destruction of renal tissue, and then remove the ureteral stone during the convalescence or as soon thereafter as possible. It is probable that a loin fistula will persist until the removal of the ureteral obstruction.

Relatively small uninfected renal stones in the kidney pelvis should be removed by pyelolithotomy, a relatively safe and easy operation when a kidney can be brought up onto the loin or even into the incision. The incision in the pelvis should be on its posterior surface and in the direction of its long axis. While the posterior surface of the pelvis is usually free of vessels, anomalies are so frequent that the operator must assure himself that the proposed line of incision is free of vessels of any considerable size. Because of this variability of the renal blood supply, one

may find in a given case that the anterior surface of the renal pelvis is the safer place to open it. The incision in the renal pelvis should be long enough to allow the removal of the stone in question without producing too much trauma. Should it be necessary to enlarge one's incision, it is better to increase it into the hilum of the kidney rather than into the upper end of the ureter. After the stone has been removed it should be examined as to whether it has any broken surfaces or as to whether it presents any facet indicative of the presence of broken fragments or of other stones. If one finds either of these conditions, one should continue his search to find and remove the broken piece or to find the remaining stone that caused the facet on the removed stone. The incision in the kidney pelvis should then be brought together with fine catgut It is especially important that one repair with care the incision into the hilum of the kidney if it has been necessary to make one. Sometimes a small oozing vessel must be controlled by a fine catgut stitch. If possible a small bit of perirenal fat should be put over one's line of incision.

I have always believed that it was a mistake to drain a kidney pelvis through a pyelotomy wound, especially by tube drainage, for the reason that stubborn fistulæ have on occasion followed this. For this reason I consider pyelotomy is not so desirable if there exists any particular degree of infection. If for any reason one finds more infection than he expected and wishes to drain a kidney on which he has done a pyelolithotomy, he should make an incision from the cortex down through into the kidney pelvis and put in a tube. Likewise if one displaces a stone from the kidney pelvis up into a calyx and cannot remove it by means of forceps I believe it is wise to open through the cortex and remove it. I find that in a few cases it is of advantage to combine a nephrostomy with a pyelolithotomy. If a kidney is bound down by adhesions or if a kidney has been operated upon previously it is oftentimes very difficult to mobilize it sufficiently to do a pyelolithotomy. The operation, however, is most useful in a considerable number of kidney stones.

Nephrolithotomy is, however, the operation that in my experience one is forced to employ in the greater number of cases of renal stone. In this operation, after the kidney has been mobilized as much as possible, even to the point of bringing it out into or onto the loin incision, one should make an incision on its convex surface just behind the midline. This should be carried down to the renal pelvis. This incision will vary from one to three inches in length, depending upon the size of the stone to be removed, and should be so placed as to bring its center over the position that the stone is felt to occupy by palpation or is seen to occupy from the *x*-ray. Various ways of opening a kidney other than by a knife have been advocated at times, such, for instance, as passing a needle threaded with wire through it longitudinally and sawing the kidney open with the idea that it causes less hemorrhage than a clean incision. I have not used any of these methods but simply cut down onto the stone with a knife. After the cortical incision has been made, a stone of moderate size can often be grasped by forceps and removed very easily. A small stone may be difficult to find. A large branching stone may require that a kidney be opened from pole to pole and the utmost care exercised in working the stone loose from the calyces and the pelvis. If on examining a stone of this type it is found that bits have broken off, the kidney should be carefully irrigated with sterile water to wash them out if possible. I personally have not used the portable *x*-ray at the time of operation although I believe that it is of a very considerable advantage in a limited number of cases. It is well to remember that if a kidney shows multiple stones one must be sure to get the full number shown and must even consider the possibility of a greater number, as I have had the experience of finding more stones than my *x*-ray called for, when one has been directly behind another. I believe that a nephrolithotomy incision should rarely be closed without drainage. I have seen a number of instances in which this was done and the kidney had to be reopened and drained because of hemorrhage. I think the best way to prevent this postoperative bleeding is to carry a relatively soft drainage tube, perhaps one-fourth inch in diameter, through into the kidney pelvis, and then bring the renal tissue together by deep catgut sutures on either side of this tube, spaced perhaps at half inch intervals. Bleeding which may have been very profuse during the time of operation usually ceases when the kidney is replaced in the loin. I feel that it is desirable to make use of a tube with a "fish tail" end in draining a kidney pelvis.

When I used tubes with lateral openings I sometimes had quite a sharp bleeding at the time I removed the tube, due to pulling off granulations that had pushed into its lateral eyes. It is sometimes necessary to drain a kidney for some little time if it has been infected; adequate drainage likewise gives a cavity that has resulted from the growth of a stone its best chance to obliterate itself. Both these measures tend to diminish the tendency to the reformation of a renal stone which, at best, is only too common.

Those patients who present a calculous pyonephrosis must be subjected to nephrectomy. In any case in which, however, there is a fair possibility that a conservative operation may succeed I believe that we should make it the rule to do this. It is perfectly true that we shall have to do a secondary nephrectomy in a certain number of these cases but in a few we shall bring about a saving of renal tissue that may add materially to the length of a patient's life.

In theory a stone that has entered a ureter should make its way to the bladder and I dare say that the great majority do, but we know a certain number do not before at least bringing about a total destruction of the corresponding kidney. My rule is that a stone in a ureter that shows any particular degree of infection should be removed, likewise a stone in a ureter that causes more than moderate pain or occasional disability had better be removed. Nor do I think we should allow a ureteral stone to remain at one place for too long a time for fear of thickening of the ureter that may lead to a real narrowing later. When one asks just what "too long a time" is, I shall have to confess my inability to define it.

When the pain of a stone low in the ureter is moderate, I am inclined to try "dilating" the ureter with one or more ureteral catheters. These I sometimes leave in place for a day or more. In some instances a stone has been passed shortly after the removal of the catheter; in other instances I have tried this maneuver without the slightest success. It has in my opinion a definite field of usefulness in the case of non-infected small stones low in a ureter. With the larger stones and especially in the infected cases I doubt the wisdom of using this procedure. One of our chief objects in the removal of a ureteral stone is to conserve the kidney as much as possible. When infection has taken place I believe the immediate surgical removal of a ureteral stone is a far safer procedure than dilatation of the ureter.

A ureteral stone may be reached at any point along the course of the ureter by a retroperitoneal incision. A ureteral stone as low as the lower border of the third lumbar vertebra may be reached easily by the oblique incision, commonly used to explore the kidney. To reach a ureteral stone below the body of the third lumbar an incision that starts a finger's breadth internal to the anterior superior spine and extends downward, parallel to the fold of the groin and about three-fourths of an inch internal to it and ends above the pubes, a modified Pfannensteil incision, gives a good exposure of the lower end of the ureter, as does a straight incision along the outer edge of the rectus muscle. In both these incisions the peritoneum is displaced inward and the ureter if dilated will be easily recognized even in the depths of the pelvis. If it is not dilated, it must be searched for at the brim of the pelvis, where it will be found crossing the external iliac artery just at the point where this vessel leaves the common iliac. The ureter at this point often adheres to the peritoneum. Once found it may be followed practically to the point where it enters the bladder wall.

In all cases but those in which a stone is impacted in the lowest part of a ureter, one should, if possible, have an *x*-ray taken just before operation. A short time ago a stone in a dilated ureter that had given a good deal of discomfort and had been located well up in my diagnostic *x*-rays was shown by a preoperative *x*-ray to be low in the ureter, from which position I removed it.

It is at times very easy unintentionally to displace upward to a point where one may have to extend his incision in order to remove a stone that is relatively low in a dilated ureter. On the other hand, it is at times most difficult to "milk" a stone that is impacted in the lower part of the ureter. up to a point where it is easier to open the ureter than is the case in the depths of the pelvis. Occasionally a curved stone forceps may be introduced through an incision in a ureter, an inch or two above an impacted stone, and the stone grasped and pulled back. In a very few instances the introduction of a probe has served to push a stone, held in the lowest part of a ureter, through into the bladder. Occasionally one may be able to isolate a ureter in its lowest part

so that he can grasp an impacted stone between his thumb and forefinger and cut down upon it. This blind procedure is to be avoided, if possible, but may occasionally be necessary. The vas deferens is in very close relation to the ureter in its lowest part and may be injured by this procedure. I have seen a mild epididymitis follow a ureterolithotomy where one had bruised the lower end of the ureter and presumably the vas as well in the removal of an impacted stone. After the removal of a stone low in a ureter, one should always make sure that he can pass a probe through into the bladder without any sensation of grating.

If after the removal of a stone an incision in a ureter can be brought together with 0 or 00 catgut, it expedites its closure. A rubber wick should be carried down to the incision in the ureter, for most of these incisions leak for a time but the rule is that they close whether they are brought together or not. When they do not close promptly an inlying ureter catheter for a few days has always brought about the desired result.

The frequency with which we see the recurrence of renal stone following operation impresses me more and more with the importance of the after-care of these patients. In this we have not alone the factor that predisposed to the original stone formation with which to cope but also certain additional factors, the result of the original stone, such, for instance, as deformities of the kidney calyces and pelvis that favor retention and persistence of infection, both probably important factors in the reformation of stone. This is a large question that merits our most careful study but one, however, that we cannot consider at this time.

SURGICAL TECHNIC IN THE TREATMENT OF URINARY LITHIASIS*

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THE Minnesota State Medical Association is most fortunate in having Dr. Arthur Chute of Boston as one of its guest speakers. He is recognized as a leader among urologists of this country, because from his vast experience in surgery he is able to demonstrate all the pertinent facts known concerning any subject in the field of urology. The paper which Dr. Chute has just read has thoroughly covered, in a few words, every point concerning the subject of "The Recognition and Treatment of Renal Lithiasis." This paper contains so much of practical value that any practitioner who has heard it will leave this room with a usable knowledge of the subject.

My discussion of surgical technic will emphasize statements made by Dr. Chute and will give you my personal reasons for suggested changes in our present surgical treatment of renal lithiasis.

The exact etiology of renal stone is not known. Certain facts concerning its formation are accepted by most surgeons. Renal infection plays a major rôle in the development of these stones and interference with drainage most surely predisposes a kidney to stone formation. Any condition which does not permit the easy passage of urine through the ureter from the pelvis of the kidney will interfere with the passage of small calculi through the ureter. The part which chemistry plays in the formation of the urine is not clearly understood. Some clinical experience points to the fact that the ingestion into the body of certain foods, and more particularly of water high in mineral salt content, is likely to cause the formation of stone. People who live in countries where animal parasites are frequent invaders of the body through food or water have frequently urinary stones. Since human kidneys cannot be the subject of experimentation in vivo, the factor or factors directly responsible for renal stone development will necessarily remain somewhat conjectural.

Since infection and interference with drainage are etiologic factors which the surgeon may control, I believe that his surgical technic must take these into consideration.

Conservatism in renal surgery is practiced by all urologists and most surgeons who have had sufficient experience to make accurate urologic diagnoses. This is particularly true when the treatment of renal calculus is considered. Wide surgical exposure is necessary. The incision must be sufficient to allow easy and thorough examination of the kidney and the renal pedicle. Anomalous blood vessels, either in the pedicle or in the lower pole of the kidney, must be recognized, so that if nephrectomy must be done or if accidents occur, the pedicle or the anomalous vessels may be clamped and bleeding controlled. For exploration of the kidney and ureter I like the loin incision of the hockey-stick type, which permits easy exploration of the upper two-fifths of the ureter. This incision may be enlarged to allow exposure of the upper three-fifths of the ureter if necessary. A midline suprapubic incision will permit exploration of the lower two-fifths of the ureter. With this type of incision, one is very much surprised at the ease with which the ureter may be mobilized at its entrance to the bladder and followed in its course through the bony pelvis.

Operations for the removal of stone from the kidney consist of incisions through the pelvis of the kidney or through the cortex. The majority of these are through the pelvis and the posterior approach is usually selected.

If merely a pelvo-lithotomy were done for the relief of stone in the kidney, many patients would not be relieved at all of their symptoms and a recurrence of stone would be frequent. Since all renal stones originate in the pelvis in a minute form, one wonders why they do not pass out of the pelvis and through the ureter while they are still small. In addition to the removal of the stone from the pelvis, the surgeon should be sure that there is nothing extra-ureteral or extra-pelvic, such as bands, anomalous blood ves-

*From the Department of Urology and Dermatology, The Nicollet Clinic of Minneapolis. Read before the Minnesota State Medical Association meeting, St. Paul, May 15, 1929.

sels, or inflammatory adhesions that immobilize the ureter and produce turns or kinks which constrict it. These are frequently found at or near the uretero-pelvic juncture. The exploration of the upper ureter and the uretero-pelvic juncture for these conditions must be made with the kidney in its usual position before it is delivered into the wound. Very frequently if the kidney is delivered outside the wound, conditions of this character may not be observed.

In addition to this exploration of the outside of the ureter and uretero-pelvic juncture just described, I believe that in every instance the incision that is made in the pelvis of the kidney for the removal of stone should be large enough to permit the surgeon to explore visually from within the uretero-pelvic juncture. We have observed many cases of narrowing at the uretero-pelvic juncture, either as the result of the continuous irritation of the stone, or of infection, or both, without external evidence of this condition.

Many pyelograms made to orient stone shadows within the kidney show a pelvic deformity at the uretero-pelvic juncture. This may consist of an over-riding of the upper ureter by the lowermost part of the pelvic shadow. In other words, the pyelogram outlines indicate that the juncture of the ureter with the pelvis is above the lowest part of the pelvis. This pelvic deformity, although not always an indication of intra- or extra-ureteral narrowing or trap formation, should cause the surgeon to thoroughly inspect the renal pelvis at its juncture with the ureter, from within as well as from without. When in doubt, a plastic operation should be done. Many of these conditions at the uretero-pelvic juncture are congenital, and therefore frequently bilateral. Since these obstructions interfere with drainage, they predispose the kidney to infection as well as to stone formation.

Two recent instances, which will be illustrated by lantern slides, will show that the patients would not have been relieved of their symptoms if an obstruction at the uretero-pelvic juncture had not been found in this manner. When obstruction in this area is found, some plastic operation should be done which will widen the area and thus improve the drainage from the kidney.

During the last few years, pyeloscopy has been developed to such an extent that it is useful in determining the function of the renal pelvis and the ureter, together with the emptying time of the renal pelvis. I am sure that if this diagnostic aid is used in conjunction with pyelography, we will find many intra- and extra-ureteral swellings or obstructions which interfere with drainage of the sodium iodide from the kidney sufficiently to lengthen the normal emptying time and make this easily visible on the screen.

Except in an emergency, where one kidney has already been operated upon, my personal opinion is that wide section of the kidney or an incision through the cortex for the removal of stone is not justified. All of these kidneys are infected, the bleeding is not easily controlled, and either infection or bleeding may make eventual removal of the kidney necessary. I have never been able to excuse myself when I have made even a small incision through the cortex of the kidney to remove a small stone. The chance that a small stone may get into the ureter is great (unless the uretero-pelvic juncture is obstructed), and the operation for its removal through the pelvis, comparatively speaking, is slight, and I therefore think that the patient should have the benefit of waiting until this may be done.

After a calculus has been removed from the upper urinary tract, that is, the renal pelvis, the kidney itself, or the ureter, thorough exploration of the uretero-pelvic juncture and the ureter should be made by means of bougies, before the incision is closed. A radiogram or fluoroscopic exploration of the kidney, outside the loin wound, should be made, to be sure that no stone fragments are allowed to remain, or that no unsuspected stone may be present. A fistula through the loin incision is frequently caused by an unsuspected stone fragment which has been pushed into the ureter.

If at the time of operation the surgeon removes a stone, explores the kidney pelvis and ureter so that he knows that drainage is complete, he then has to face the question of the removal of infection already in the kidney and the control of quiescent infection. Since this is a major factor in the production of renal stones, I believe that every patient should be thoroughly studied for focal infections. To control the remaining infection in the renal pelvis or upper ureter, my patients are advised to submit to regular pelvic lavage for at least six months following the removal of stone. In every instance, I insist that all foci of infection be removed.

PERVERSIONS OF THE FUNCTION OF THE DIAPHRAGM*

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GENERALLY, the diaphragm is considered as a muscle important for its share in the phenomenon of respiration and as a partition between the abdomen and chest. Various phases of its function, normal and perverted, have great interest for the surgeon, and my own interest has been stimulated by a series of studies now being made in our clinic, a preliminary report of which has been made to the recent meeting of the American Association for Thoracic Surgery. No attempt will be made to write a complete report upon the diaphragm, the recent papers of Lemon, Campbell, Funk, and, further back, those of Hoover, being quite informative.

The diaphragm develops very early in embryonic life, arises toward the head and migrates caudally: Its ventral pericardial portion is formed from the septum transversum, its lateral portion from the pleuro-peritoneal membrane and parts derived from the body wall, and the medial dorsal portion from the primitive mesentery. The derivation from the body wall has been in dispute because of the uncertainty regarding the innervation by the intercostal nerves.

The comparative anatomy of the diaphragm has received considerable attention, particularly from Keith, Jones and Kingsley, to mention but a few. Lemon concludes that "it would appear from the study of comparative anatomy that structures similar to the diaphragm play varying parts in respiration, acting as expiratory, as inspiratory and at other times as both expiratory and inspiratory organs. Without doubt higher forms of animals, possessing true lungs and equipped with musculature which provides efficient respiration, breathe without the aid of a diaphragm. It is likewise true that a perfected diaphragm coincides with a mechanism developed to support high intracelomic pressure. All of these facts lead one to believe that the providing of pressure is the chief and primary function of the diaphragm and that its purpose as a partition is to allow the increased pressure to act on abdominal viscera permitting them to discharge their contents (fecal matter, urine and offspring), while at the same time the heart and lungs are spared the effects of the same pressure. It would appear that animals such as the dog can maintain respiratory efficiency without impairment when half of the diaphragm has been paralyzed and even when both sides are functionless and, in fact, a hindrance to respiration. In such animals the diaphragm does not compare in functional importance with the heart."

As a result of hundreds of observations after unilateral phrenic exeresis, it is apparent that man can live comfortably with hemi-paralysis of the diaphragm but I am not impressed by the reports of cases of bilateral phrenicotomy for persistent singultus as not causing noteworthy respiratory disturbance. In the past few years I have frozen the phrenics on both sides five times and, while the singultus was cured, the diaphragm *did* move, as seen by the fluoroscope shortly after operation, showing evidence of the transmission of motor impulses below the level of freezing by way of the accessory derivation.

Lemon found that a dog can live comfortably without diaphragmatic movement but the evidence regarding man is rather scanty. Neuhofer[2] has reported the case of a boy eight years old who had phrenicotomy done on both sides. Over three years later the diaphragm was found still practically immovable. The boy had developed normally, the thoracic respiration answering all purposes. However, vital capacity, restriction of external respiration and impaired competency are bound to result. Epstein[3] believes that the hypoventilation will predispose the patient to respiratory infections.

The anatomy of the diaphragm in man is well known and does not need detailed description. It has been spoken of as the most important muscle of the body, next to the heart. This is no doubt true under normal conditions, Lee[1] and his associates, of Columbia University, having found under various experimental conditions that it is vastly superior, physiologically, to other muscles studied.

"The peripheral edges of the diaphragm are

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attached behind to the lumbar vertebræ, laterally to the lower edges of the six lower ribs and their cartilages, and in front to the tip of the ensiform cartilage. The fibers converge to enter the central tendon, and the lateral sheets are pressed upward by the intra-abdominal positive and intrathoracic negative pressures, so that they form a dome-shaped vault, with the liver in the right side and the stomach and the spleen on the left."

"Because of its attachments to the lower six ribs, the contraction of the diaphragm tends to pull the margins of the ribs towards the median line, but under normal conditions this movement is opposed by the action of the external intercostals in raising the ribs and expanding the horizontal diameters of the thorax, and by the lower vertebral muscles, which fix the position of the lower ribs." (MacLeod.)

Hoover[4, 5] has given the greatest impetus to the study of the movements of the diaphragm and its significance in disease. He called attention to the fact that clinicians concerned themselves mostly with the density of the lung and some adventitious sounds and neglected the study of volume and extensibility of the lung. He studied particularly the movements of the costal margin and showed that phrenic contraction tended to narrow the subcostal angle, and maintain or enlarge the longitudinal diameter of the thorax. Variations in respiratory movements of the lower end of the sternum, subcostal angle and costal borders from the ninth rib to the subcostal angle, the epigastrium and hypochondria all depend on the higher or lower positions of the diaphragm. The diaphragm is correlated with the scaleni and intercostal muscles but antagonistic in its resistance to lessening of the longitudinal diameter of the thorax. The movement of the costal margins expresses accurately the balance between the intercostal muscles and the diaphragm in their contest for mastery of the costal margins. Hoover mentions a case in which physical signs suggested thoracic empyema following acute appendicitis. There was dullness at the base, diminished tactile fremitus, diminution of the intensity of the respiratory sound with elevation in pitch. But, the costal border of the affected side had a greater lateral excursion than the sound side. This meant a loss of the normal antagonism of the diaphragm to the action of the scaleni and intercostals of the affected side, hence an upward displacement of the diaphragm and a subphrenic abscess.

The surgical anatomy of the diaphragm has been ingeniously applied by Mosher[6] and by Jackson.

From various studies Mosher found that the diaphragm formed a cone about the terminal portion of the esophagus acting as a sleeve. The left crus largely and the right crus to a lesser extent exert pressure on the esophagus at the hiatus and steady it. In cases of visceroptosis the diaphragm has descended and the cone is flattened, thus mobilizing the terminal portion of the esophagus and tending to cause dilatation and cardiospasm. Jackson draws the analogy of the laboratory pinchcock on the rubber tube to the pinchcock action of the crura and explains some of the cases of non-organic stenosis to accentuation of this "pinchcock."

One other effect of the diaphragm should be considered here, namely, the effect on the blood pressure. "Because of the increase in the intra-abdominal pressure the viscera are crowded against the inferior vena cava, blood is forced into the thorax and the immediate effect is that of raising the blood pressure temporarily. Before inspiration is complete the lessened flow tends to decrease the blood pressure. Abdominal respiration produces an initial rise succeeded by a fall in blood pressure during inspiration and an initial fall followed by a rise in pressure during expiration. These results are opposed by the effect on pressure caused by thoracic breathing in which the pressure falls and later rises during inspiration. The effect of combined breathing is the algebraic sum of these opposing influences." (Lemon.)

The nerve supply of the diaphragm is exceedingly important for obvious reasons. Curiously it is not uniformly stated in different anatomies. According to Gray, "the diaphragm is supplied by the phrenic and intercostal nerves."

Deaver[2] states that the nerve supply comes "from the phrenic nerves and lower five or six intercostal nerves. Small filaments are also sent to the diaphragm from the diaphragmatic *(phrenic)* plexuses, which are derived from the semilunar *(celiac)* ganglia and solar *(celiac)* plexus. It should be remembered that section of the phrenic nerve in the neck does not paralyze the diaphragm. The accessory phrenic which comes from the fifth cervical does not join the main nerve until this nerve has passed below the clavicle."

According to Morris, the nerve supply of the diaphragm comes "from the phrenic nerves, one of which arises on each side from the third to the fifth cervical nerves. Each nerve penetrates the diaphragm lateral to the central tendon and breaks up into an extensive plexus on the inferior surface of the muscle. Some of the lower intercostal nerves also contribute to the sensory innervation of the margin of the muscle and possibly also slightly to the motor innervation."

Piersol and also Cunningham attribute the nerve supply of the diaphragm entirely to the phrenic nerves. According to Piersol, innervation is derived "from the third, fourth, and sometimes the fifth cervical nerves, by the phrenic nerves."

Cunningham states that "the diaphragm receives its chief, if not its entire, motor supply from the *phrenic nerves* (C.3.4.5.). It is innervated also by the diaphragmatic plexus of the sympathetic, and is sometimes said to receive fibres from the lower thoracic nerves."

The bulk of independent investigation supports the view that almost the entire motor innervation of the diaphragm comes through the phrenic nerve. Felix in 1923 concluded from his experiments and clinical studies that the motor nerve supply is derived from the phrenic alone. A few motor fibres reach the diaphragm from the twelfth intercostal nerve, but they are not of importance. The muscular tonus of the diaphragm persists only as long as the motor nerve supply is intact. The expansion of the diaphragm persisting after complete motor paralysis of the diaphragm is not due to muscular tonus governed by some special nerve supply but to the action of outside forces—the traction of the lungs, the pressure of the abdominal organs, and the traction of one-half of the diaphragm on the other in cases of unilateral phrenicotomy or blocking of the phrenic nerve.

Schlaepfer in 1923 reported that in experiments on dogs he found that resection of the phrenic nerve on one side caused complete atrophy of the diaphragm on that side. Faradic stimulation of the phrenic nerve on one side caused a prompt contraction of the same side of the diaphragm. Stimulation of the intercostals, the vagus and the anterior roots of the lower thoracic and upper lumbar nerves caused no movement of the diaphragm. Schlaepfer concluded that the phrenic nerve is the only motor nerve for the diaphragm—at least in dogs—and, according to clinical observation, also in man.

Meisezahl in 1925 reported a case in which excision of 34 cm. of the phrenic nerve on the left side was followed by loss of movement and elevation of the diaphragm. The patient died a little over a year later, and at autopsy it was found that the left side of the diaphragm showed complete and uniform muscular atrophy. This finding would seem to indicate that the phrenic is the only motor nerve supplying the diaphragm. Confirmation of this result is found in the experiment published by Schlaepfer.[7]

Through a transpleural approach, the left phrenic nerve in a dog was resected for a length of 1 cm. in its passage over the pericardium, and the experiment terminated two years after this operation. At necropsy the whole left diaphragm formed a loose fibrous membrane contrasting with the muscular right side. As demonstrated by photographs, the muscular atrophy was complete in the lumbar and esophageal portion of the paralyzed diaphragm. A few atrophic muscle fibres were found next to the midline on the paralyzed side in the sternal portion and anterior to the esophagus.

According to Kure, "the diaphragm gets its nervous supply mainly from the phrenic nerve. Besides this, it has the sympathetic innervation coming from the celiac ganglion." The phrenic nerve, he continues, contains non-medullated fibers derived from the cervical sympathetic, especially the stellate ganglion. Thus the sympathetic nerve supply of the diaphragm can be entirely eradicated without impairing the somatic innervation if the stellate ganglion and the abdominal sympathetic are extirpated. On the other hand, the cerebrospinal innervation can be eradicated by severing the roots of the phrenic nerve. In experiments on monkeys, Kure in 1928 found that removal of either the cerebrospinal roots or the sympathetic fibers of the phrenic nerve causes only temporary and slight decrease in the tonicity of the diaphragm. When both are eradicated, the decrease in tonicity is marked and permanent. He concluded, therefore, that both the cerebrospinal roots and the sympathetic fibers participate in maintaining the tonicity of the diaphragm. If one is deficient, the other compensates for it in maintaining this tonicity.

Sensory nerve supply.—The central portions of the pleural and peritoneal surfaces of the dia-

phragm receive their sensory nerve supply from the phrenic, the outer margins from the lower six intercostals. After some ingenious experiments Capps and Coleman[8] conclude that "the peritoneum covering the diaphragm is devoid of pressure sense as applied by light contact or stroking of a beaded wire point. But to strong pressure with a beaded point or light contact with a rough point it is acutely responsive to the sense of pain." Further, they found that "the localization of pain from stimulation of the diaphragmatic peritoneum is never in the diaphragm itself. It is always referred to some distant part. Stimulation of the outer margin causes diffuse pain over the lower costal region and subcostal abdominal wall. Stimulation of the central portion produces pain over a sharply limited point somewhere along the trapezius ridge. These impulses are doubtless carried by afferent fibers of the phrenic nerve to the cervical cord and thence referred to the neck by the sensitized cutaneous nerves of the fourth cervical segment. This pain has not been observed along the course of the phrenic nerve itself."

The diaphragm may be involved in various inflammatory affections, either acute or chronic, subphrenic or supraphrenic. It may be reflexly affected as after operation or spasmodically affected as in hiccough. Its function may be deliberately altered by section of the phrenic nerve. It may be deficient anatomically and allow diaphragmatic hernia to occur. The movements of the diaphragm may be accurately determined by observing Litten's phenomenon, by palpating the costal margins by the method of Hoover or by means of the roentgen ray.

Pain from the affected diaphragm may radiate downwards to the abdomen or upwards to the neck. We are all familiar with the occasional case of pneumonia especially in children in which abdominal pain and rigidity have suggested acute appendicitis and at times the differential diagnosis may be most difficult. A knowledge of the external manifestations of perverted diaphragmatic movement is very helpful in these cases, the evidence of a costal type of breathing, absence of Litten's sign, limitation of movement of the costal margin on the affected side. Sale[9] made an interesting study of the diaphragmatic movements after the onset of acute abdominal symptoms suggesting appendicitis. Flattening of the movement of the right diaphragm was noted in sixteen cases diagnosed and found at operation to be acute appendicitis; in these other cases the sign was positive but the clinical signs obscure; later operation revealed acute appendicitis in all. A negative sign was corroborated in three cases by operation disclosing a normal appendix and in three cases by the clinical recovery of the patient. He quotes Erlanger as stating that as there are no inhibitory fibers in the phrenic, the diaphragm may be mobilized simply in a mechanical fashion through rigidity of the abdominal wall.

Capps[10] made some interesting experimental observations on this point. In cases of pleural effusion he found that pressure excited by a probe on the outer edge of the diaphragm produced painful sensations referable to the lower portions of the thorax, flank or abdomen.

Pain in the shoulder, at the outer border of the trapezius, is well known as a phenomenon attending certain lesions below and involving the diaphragm and here again Capps has added to our exact knowledge. He was able to produce a pneumoperitoneum in patients and under the fluoroscopic screen watch a beaded wire as it was pressed against the diaphragm. Pain in the neck was caused by strong pressure of the wire on the central portions of the diaphragm and in the lower costal and subcostal areas by pressure over the outer portions.

It is well known that subdiaphragmatic abscess produces elevation of the diaphragm and pericardial effusion depresses it. These diseases will not be discussed. In 1916 Pryor[11] called attention to the immobility of the diaphragm following pleural exudates, such as empyema, pleurisy with effusion, and pulmonary tuberculosis with effusion. In about 60 per cent one-half of the diaphragm was completely immobile and in about 20 per cent the excursion was more or less restricted. The observations were made from one month to nineteen years after operation. He seemed to conclude that this condition had resulted "from a gradual paralysis of the phrenic nerve, induced in its course through the mediastinum along the pericardium and phrenico-pericardial ligament to the diaphragm." This region constitutes the point of greatest lateral pressure accompanied by displacement of the heart. The pericardium is held in position below by the diaphragmatic attachment and above by the cervical fascias, the arrangement being much like that of a punching bag. As the heart is pushed and

twisted to the opposite side, the drag and the stretch are applied mostly to the diaphragmatic ligament.

In this connection an interesting study on the conduction of impulses through regions of block of the phrenic nerve by pressure were published a few years ago by Briscoe.[12] She showed that a weight of 400 gm. applied to 4 mm. of nerve will block in 10 minutes and a weight of 80 gm. in one and a half hours. "Since the fibrous tissue or new growth may exert pressure over periods of weeks or months, it may well be that such pathological conditions may block conduction with pressures considerably smaller than those above quoted."

Middleton[13] studied a group of patients to determine the effect of costodiaphragmatic adhesions upon the respiratory function. He concludes from his study that costophrenic synechiæ embarrass respiration to the extent of an average reduction of 32 per cent in vital capacity. This percentage reduction will be favorably influenced by other factors and thus the actual uncompensated decrease greater. He further remarks that the normal diaphragmatic contraction must account for at least 60 per cent of the respiratory interchange in deep breathing. Pryor's conclusion of an actual paralysis of the diaphragm was disproved by the finding of movements of the costal margin toward the midline or no movement whatever upon inspiration.

Matson[14] calls attention to some observations made by him which show that "tenting" of the diaphragm does not necessarily indicate the presence of pleuro-diaphragmatic adhesions. He believes that the "tenting" may be a physical phenomenon brought about by two factors: (a) "During inspiration the diminished elasticity or rigidity of the lower bronchial branches prevents descent of that area of the lung base in immediate relationship to the bronchus involved; thus a dimple effect is produced on the surface of the base of the lung; (b) the diaphragm being molded to the base of the lung by a negative intrathoracic pressure is held by suction in the dimple above referred to, producing a tented appearance (pseudo-adhesion)."

Hiccough is a clonic spasm of the diaphragm, the impulse coming through the phrenic nerves, and occurs in exhausting illness, after surgical operations, from a distended abdomen, from acute indigestion, from reflex causes, as a functional manifestation, etc. An interesting type of hiccough is that seen in the affection commonly called "epidemic hiccough," which, however, seems to have occurred coincidentally with epidemic influenza or epidemics of myoclonic encephalitis. I have seen three cases of post-encephalitic tic of the diaphragm and in all the diaphragmatic spasm was cured by freezing the phrenic nerve in the neck.

The first case was reported in 1925 by Gamble, Pepper and myself[15] and occurred in a man thirty-eight years of age who in November, 1924, had acute epidemic encephalitis. In January, 1925, he first noticed the disturbances of respiration and two months later when we first saw him he was suffering from "dyspnea" as expressed by rapid chest movements following the tic or spasm of the diaphragm and occurring between 60 and 90 times a minute.

"When, under the fluoroscope, the patient was instructed to take a deep breath, the diaphragm could be seen moving in inspiration and expiration, with the more frequent minor contractions of the tic superadded to the larger excursion. When the breathing was not voluntarily controlled, the only motion of the diaphragm was the tic."

Blood chemistry studies were made by Dr. J. H. Austin and he found a shift of the reaction of the blood toward the alkaline side. "These studies confirmed the view that there was overventilation going on as a result of the tic with the synchronous and rapid respiratory movements. The respiratory center was not receiving normal stimuli to respiration and was not sending out strong rhythmic impulses. It seemed possible that there was no actual damage of the respiratory center itself, as might be feared after encephalitis (post-encephalitic respiratory syndrome), but that the respiratory disturbance was wholly secondary to the overventilation induced by the diaphragmatic tic. If this were so, an abolishment of the tic should restore the breathing to normal." Accordingly I exposed and froze the phrenic nerves on both sides and upon recovering from the anesthetic the patient expressed his relief from respiratory distress. "Tracings taken by Dr. Gamble now showed normal respiratory movements, and in the fluoroscope the diaphragmatic movements could not be distinguished from those of a normal person with a small excursion. No tic could be observed. The breath-

ing was apparently normal in every way. The respiratory rate was never rapid. When the breath was held the chest was entirely quiet and none of the jerky movements, which the tic had formerly caused, could be seen. There was no dyspnea or discomfort after climbing three flights of stairs in thirty seconds." The patient was last seen 3 years and 9 months after the operation and had remained well.

The second case occurred in a young girl sixteen years of age and presented similar phenomena except that the post-encephalitic manifestations were more pronounced. She also was relieved by phrenic nerve freezing. The third case presented an unusual feature. The patient was a boy, 22 years old, whose history dated back to November, 1924, and the symptoms were quite vague but suggestive of mild encephalitis. In March, 1925, he developed the dyspnea, which was temporarily relieved by drinking water. As the hyperpnea increased the water drinking increased until at the time when we first saw him, in November, 1927, he was drinking from thirteen to fifteen quarts of water daily. Under the fluoroscope the diaphragmatic movement was normal, the excessive movement not apparent and no evidence of flutter. The phrenic nerves were frozen and thereafter the patient was unable to develop hyperpnea although the polydipsia persisted. After hard work he was reduced to an allowance of 3,500 c.c. of water each day and two months later was still well and trying hard to keep to the allowance. A few days after operation the fluoroscope showed a diaphragmatic excursion of 7 cm. on the right side and 5 cm. on the left.

A case, similar to my first case, was reported by Dowman[16] in 1927 and he quotes Marie as classifying these phenomena into: (1) disturbances of respiration proper, as tachypnea and bradypnea; (2) spasmodic cough, and (3) respiratory tics. Another case has been described by Edie[17] but the phrenics were not blocked and it is stated that the patient received benefit subjectively from luminal.

It is not necessary to dilate upon the treatment of hiccough. In 1921 DeBuen[18] enumerated twenty-three physio-mechanical methods, over thirty drugs and other procedures, all of which had merit. However, there are certain intractable cases, and I have had four such, in which all treatments have failed and direct attack upon the phrenics is justifiable. Goetze[19] in 1920 was the first to suggest injection of the phrenic nerve with novocain for the relief of hiccough. He injected 20 c.c. of a one per cent solution around the nerve and got relief. Fischer in 1923 advised freezing the nerve.

Phrenic nerve interruption as a means of obtaining therapeutic collapse was first proposed by Stuertz in 1911. He practised a simple division. In 1922 Felix proposed the method of radical extirpation or phrenic exeresis which is so commonly done at the present time. The indications for this operation and the discussion of its results have no place in this paper.

The effect of phrenic section on the diaphragm is that of a practically complete paralysis of the side operated upon with ascent of the dome of the diaphragm for a distance of from 4 to 8 cm. on the right and 2 to 6 cm. on the left side. The clinical signs it gives rise to are, "indrawing of the epigastrium with a certain loss of resistance of the abdominal muscles, the affected half of the thorax makes greater excursions, the ribs run less obliquely, the intercostal spaces are broader and the breast stands high on the side which is affected. Litten's sign is absent" (Campbell). Unless the diaphragm is fixed by adhesions or there is a basal pleural effusion, paradoxical movements will be observed. This means that with the downward thrust of the intact diaphragm, intra-abdominal pressure will push the paralyzed side upwards. The affected diaphragm atrophies and eventually becomes a mere membrane. The operation is invaluable in certain types of pulmonary tuberculosis and often useful in bronchiectasis and abscess of the lung.

Eventration of the diaphragm apparently was described first by Petit, who died in 1750 and whose writings were first collected and published in 1774 by Lesue.

Morison[20] states that all of the cases recorded have been on the left side with the exception of one by Eppinger (1911). Morison reports six cases observed by roentgen examination as diagnostic. He notes: (1) the appearance of the upper boundary of the dome as a bow line; (2) the in sac of the stomach in the dome; (3) lung tissue seen through the dome; (4) rotation of the heart; (5) deformity of the stomach, presentation of two sacs, the upper spilling forward into the lower; (6) paradoxical movements. It is interesting to record that in his first case

an operation was performed by Morley, who exposed the diaphragm by the thoracic route. It was incised and then partly excised so as "to bring down its level" and reduce the distortion of the stomach. Three years later the patient was well and suffered no inconvenience. Microscopic examination of the piece of diaphragm removed showed no sign of muscle fibers.

Hernia of the diaphragm is well known and the literature contains many papers bearing on its etiology and treatment. Some difference of opinion has arisen among surgeons as to the best method of approach, through the thorax or abdomen. In 1921 Baker[21] advocated freezing the left phrenic nerve preparatory to radical operation for hernia. He used the thoracic approach. Prompted by Harrington's paper,[22] I recently repaired this hernia through an abdominal approach. With preliminary phrenic nerve freezing and spinal anesthesia the diaphragm was so relaxed that suturing was as easily accomplished as an ordinary skin suture. In the same paper Harrington advocates phrenicotomy as a palliative procedure in certain cases of diaphragmatic hernia in which radical surgical procedures are contra-indicated. Increased intra-abdominal pressure primarily forces the stomach into the thorax, where it is maintained by the suction of the negative pressure in the thorax and the pumping action of the diaphragm. This muscle is then put into spasmodic contraction and cardiospasm or severe shoulder pains are produced. Phrenicotomy checks the pumping action of the diaphragm and by preventing spasmodic contraction relieves pain. The paradoxical movements of the hemiparalyzed diaphragm may lessen the amount of herniated contents.

The diaphragm may be affected by phrenic nerve involvement in the mediastinum from tumor or inflammatory affections. Morison reported nine such cases in which a high diaphragm was found. Necropsy in two of the cases revealed the phrenic nerve involved and some degeneration of the muscle fibers of the diaphragm and he therefore suggests the possibility of an axillary innervation of the diaphragm. However, we might also apply a suggestion of Briscoe that there might be a breaking through of the nerve block by stronger respiratory impulses.

Injury of the brachial plexus in the newborn may involve the phrenic and result in paralysis of the diaphragm. This was first recognized by Kofferath.[23] Dyson[24] has recently reported a case, which was particularly interesting by reason of the paralysis involving the right diaphragm with marked paradoxical movement and by the fact that there was no evidence of other nerve injury. No attempt at nerve repair was advised.

An interesting phase of perversion of diaphragmatic function is that concerning the relation of the diaphragm to hypoventilation of the lung after operations, especially those on the upper abdomen.

Lemon studied the effect of abdominal incisions and major surgical operations on the upper part of the abdomen to note whether the operation caused fixation of the diaphragm, thus contributing to the prevalence of postoperative pulmonary complications. Dogs were used for the experiments and in none was any fixation observed, the excursion being normal and equal to that before operation. Lemon speculates regarding the diaphragmatic movement in man under similar circumstances and states that "the low vital capacity in man is more an expression of abdominal pain preventing resistance to diaphragmatic contraction than to any loss of power in the efficiency of the diaphragm itself." Churchill and McNeil[25] studied the vital capacities in man, as affected by operations on the abdomen. The greatest reduction followed operations on the gall bladder, those on the appendix and for hernia being less marked, and non-abdominal cases not influenced. They believed that the splinting of the muscles is the chief causal factor concerned in the marked reduction of vital capacity, tight abdominal dressings bearing an important part, and posture, distention, fatigue, medication, etc., playing roles of varying importance.

In view of the greater incidence of postoperative pulmonary complications after operations on the stomach, gall bladder, spleen, etc., I conceived the idea that the disease itself was responsible for loss of power in the diaphragm and that such hypofunction was the immediate cause, aspiration into the lungs and bronchi as shown by Lemon[26] being the contributary cause of the pulmonary complication. This was in contravention to the nearly accepted opinion that emboli were the most common excitus of the postoperative pulmonary complication. We were also aware of the statement made by Lee[27] that collapse of the lung in varying degrees always fol-

lows any operative procedure, inhibiting normal respiratory movement by pain or posture.

We studied a group of twenty-five patients who were operated upon for upper abdominal lesions. The actual work was done by one of our Fellows, Dr. R. H. Overholt, and Dr. E. P. Pendergrass supervised the roentgenographic part. Pulmonary symptoms and signs were noted, chest measurements made, vital capacity estimated, and the diaphragmatic movements recorded. At this time I will discuss the latter aspect only. We did not find any evidence of disturbed diaphragmatic function prior to operation, the original starting point of the study. We did find that in nineteen out of twenty-five cases (76 per cent) distinct hypoventilation occurred.

Measurements of the chest in fifteen cases showed a marked reduction in the expansion, from 4 to 5 cm. being the usual finding. Elevation of the diaphragm was seen in all cases, most marked on the right side and especially so in two patients who later developed the signs of massive atelectasis. The maximal excursion of the diaphragm was reduced after operation to a third of its preoperative range of movement. The tidal fluctuations were diminished about one-half.

It was also observed that the position of the tidal fluctuation of the diaphragm varied in regard to its position in the maximum range of excursion. Before operation the tidal movements took place in the lower third of the possible range noted during forced breathing. However, after operation the tidal fluctuations occurred in the upper third of the possible range of movement of the diaphragm.

The comparison of the roentgenograms made before and after operation was perhaps the most striking of all of the observations made in this study. In all of the cases a definite diminution in chest volume was obvious. There was a narrowing of the inter-rib spaces and an elevation of the diaphragmatic domes. The lung markings in only six of the cases were clear. In sixteen of the cases there was a marked intensification of the trunk shadows which produced a triangular area of density in both lower lobes. On the right the cardio-phrenic angle was obliterated. On the left the markings were more or less obscured by the cardiac silhouette. In some of the roentgenograms there are varying degrees of haziness or mottling throughout the entire lung fields.

In the lateral views the appearance was just as striking. The marked reduction in chest volume was clearly evident. The costophrenic angles posteriorly were completely obliterated. The marked diaphragmatic elevation obscures the long detail in the lower lobes. Air can be seen beneath the diaphragm in many of the films. Roentgenograms made preoperatively during full expiration gave similar appearances to the first postoperative day examinations—intensifications of the trunk shadows and alteration in the size of the aerated lung.

The fact that there is hypoventilation of the lungs following upper abdominal operations has been alluded to and commented upon by many writers. However, in this study, the various physical measurements of the respiratory movements demonstrate more concretely the extent of this postoperative depression in pulmonary ventilation. The reduction of 79 per cent in chest expansion and the limitation of 66 per cent of the diaphragmatic excursions would indicate that a great portion of the reserve of the external respiratory forces was lost for a variable period of time following operation. The constant high position of the diaphragm with the tidal fluctuations occurring in the upper third of its possible range of motion illustrates the mechanical disadvantage imposed on the diaphragm after upper abdominal operations.

One of the most interesting and important observations which we were able to make was that the appearance of the base of the lung in hypoventilation presented an appearance similar to that previously diagnosed as a true inflammatory pulmonary lesion, such as a bronchopneumonia, a pneumonitis or a lobular atelectasis. This was checked by Dr. Overholt in an ingenious way. Roentgenograms made before operation of the expiratory phase in which of course there is pulmonary compression, gave a similar appearance to that seen after operation during full inspiration, except that the latter was more marked because the hypoventilation is maintained over a period of time lasting several hours after operation.

There is much opportunity for speculation in these findings but we have reserved this for our other contributions. It is well known that during depressed respiration and particularly when breathing is shallow hypoventilation occurs. Occlusion by bronchial secretions may thus lead to atelectasis. The point is not generally brought

out that the entire respiratory act is depressed, thus lessening the bronchial lumen and favoring the obstruction. A number of our patients were operated upon under spinal anesthesia and these also showed the diaphragm phenomenon described, but I have noted that in these respiration was also depressed. The causal relation of asphyxia to pulmonary congestion no doubt is linked with hypoventilation. At the present time I am under the impression that the irritation of the vagus by the operative manipulations is partly responsible for the depression of the respiratory act. The question of the sympathetic effect on the tonus of the diaphragm needs further study. Certainly the high position of the diaphragm cannot be due to phrenic nerve inhibition. We might think of an increased resistance of the abdominal musculature against the downward thrust of the diaphragm as a factor but clinical observation does not demonstrate that this often occurs.

It will thus be seen that a study of the various phases of diaphragmatic function is worth while and at any rate it stimulates a better understanding of the reasons for the various perversions of its function.

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HERNIA OF THE DIAPHRAGM THROUGH THE ESOPHAGEAL HIATUS WITH REPORT OF NINETEEN CASES*

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EXTENSIVE discussion of the types and classifications of diaphragmatic hernia may be found in medical and surgical textbooks and in numerous papers on this subject. We will therefore not enter into this in detail. The present discussion limits itself to herniation through the esophageal hiatus, which is no doubt the most frequent site of diaphragmatic hernia.

Hernia through the esophageal hiatus has been considered a comparatively rare condition until recently. Schwalbe in 1900 considered it to be very uncommon. Keith in 1910 collected thirty-four cases of diaphragmatic hernia from various pathological collections in London, and only one of these was through the esophageal hiatus. In a monograph in 1911, Eppinger made a very complete review of the literature of diaphragmatic hernia. He collected 635 cases of true and false hernia, and of these eleven were of the para-esophageal type. In 1920, Quenus collected 116 operated cases of diaphragmatic hernia, and only two of these were of the para-esophageal type. In 1924, Carman and Fineman of the Mayo Clinic reported seventeen cases of hernia discovered roentgenologically, and only one of these was through the esophageal hiatus.

More recent work by Åkerlund presents a different angle on this subject. His review of the literature is very complete. He found reports of at least sixty cases of this type. Over a period of five years he collected twenty-four cases himself, sixteen of which he observed personally, while the others were observed by Forssell, Panner, and Simon. Åkerlund now reports that he sees on the average one of these cases per month. He considers this type to be the most common hernia of the diaphragm. A recent report by Healy, presenting fifty-three cases, and our own experience, substantiate Åkerlund's opinion of the frequency of this condition.

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Åkerlund distinguishes three types of hiatus hernia, as follows:

1. Hernia through the hiatus esophageus with congenital shortening of the esophagus, thus making a reposition impossible.

2. Para-esophageal hernia in which the herniated sac lies lateral to the otherwise normal esophagus, which is of normal length and in normal position.

3. Hernia in which the esophagus is not shortened, but in which its distal end constitutes a portion of the contents of the hernia sac.

Åkerlund found the first type to be comparatively rare and classified only nine cases from the literature into this group. He found that these cases tend to show a large portion of the cardia of the stomach in the posterior mediastinum, with bulging into either pleural cavity, more commonly the right. This type is definitely congenital in origin and is non-reducible, owing to the shortening of the esophagus. LeWald calls this type "thoracic stomach" and reports two cases. He considers this to be a congenital anomaly, and that it should not be classified as a hernia. Carman and Fineman's case is also of this type.

The second type is a more common type of hernia. In this type the hernial sac lies lateral to the esophagus, either to the right or left, and occasionally anterior or posterior. The esophagus has a normal linear development. Eppinger thinks that this type is due to a congenital weakness in the muscular ring of the hiatus, and that the hernia develops in the course of extra-uterine life. This type of hernial sac may contain either the cardia or the lower portion of the stomach, as well as the duodenum, other loops of bowel, or the omentum. None of Åkerlund's cases come under this type, but Eppinger reported two cases, and Schwalbe, Manenkow and Legry each report one case under this group. One of our cases (Case 1) belongs to this group.

The third type is closely interrelated with the

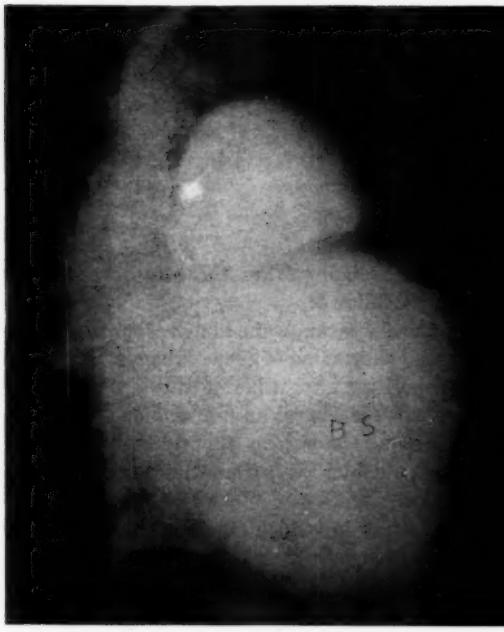


Fig. 1, Case 1. Hiatus hernia with gastric antrum and pylorus in hernial sac.

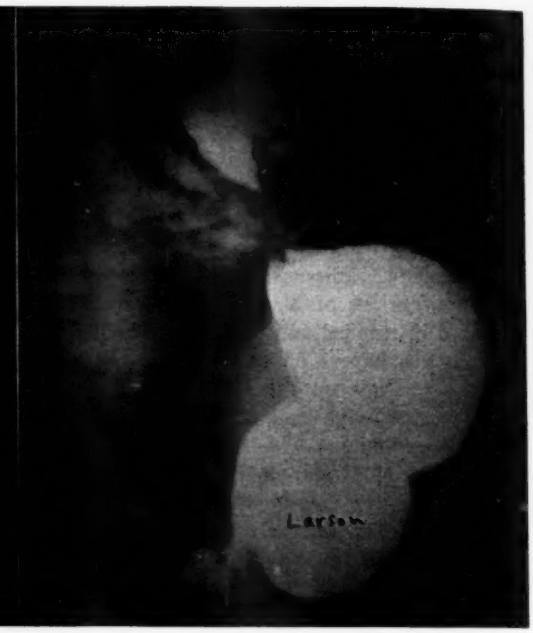
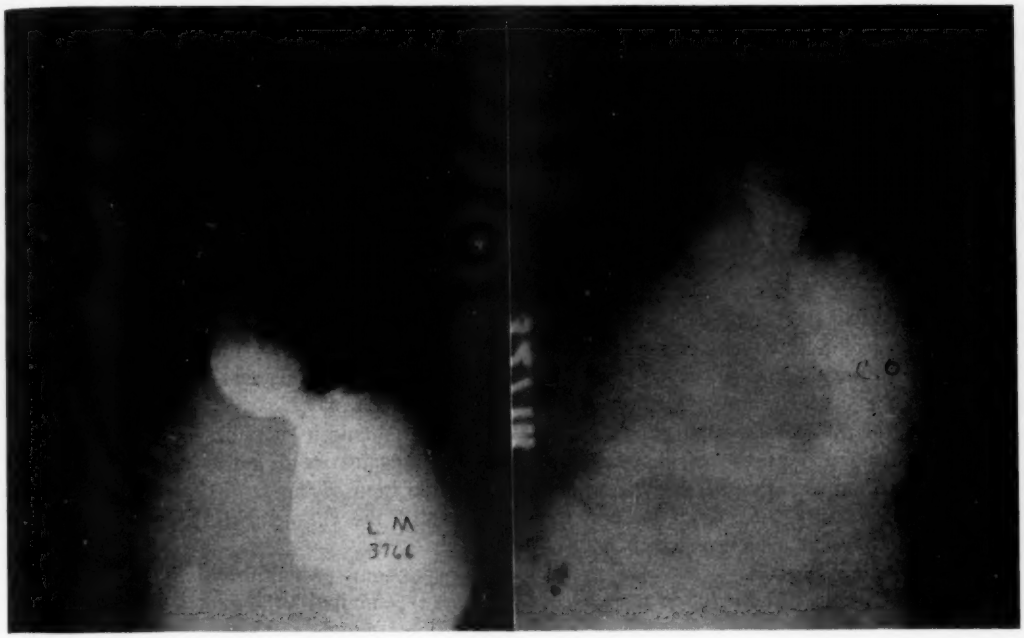


Fig. 2, Case 4. Hiatus hernia with large portion of cardiac end of stomach in hernial sac.

second, varying from the latter in that the distal end of the esophagus lies entirely above the diaphragm, within the hernial sac, where it describes a somewhat tortuous course and shows some dilatation. This group comprises the majority of the reported cases, including all of Åkerlund's cases and all but one of our series. The size of the hernial sac in this group is usually smaller than in the first group, and usually produces no material displacement of the heart.

Many herniæ of the esophageal hiatus are probably purely congenital in origin, being on the basis of a persistent congenital sac, or due to an anomalous condition of the muscular ring. Some may be purely on an acquired basis, the factors being a reduction of the subperitoneal fat, an atrophy of the muscular ring, or stretching of the ring by increased intra-abdominal pressure. One of our cases (Case 1) appears to be definitely related to childbirth, while nine of our cases were in women who had had multiple pregnancies. One was found to have large uterine myomata. One had a very large ovarian cyst. Five cases were associated with a marked increase in intra-abdominal pressure due to ascites and extensive malignancy. Obesity was present in five, and constipation in two cases. This close relationship of many cases to increased intra-abdominal pressure leads us to believe that this is an important etiologic factor. One of our patients had a marked deformity of the dorsal spine and thoracic cage from an old tuberculous spondylitis. Only one case gave a history of severe trauma, but the onset of the symptoms occurred nineteen years after the injury and was related to ascites, so that the injury was probably not a factor. Storonoff and Soresi each report a case where direct trauma to the hiatus was the etiologic factor.

Roentgenologic studies.—Most herniæ through the esophageal hiatus are found accidentally during the course of a roentgenologic study of the gastro-intestinal tract. The diagnosis is rarely made clinically. In several of our cases our attention was directed to this region during the preliminary fluoroscopic study of the chest, namely by a displacement of the heart to the right and by the presence of a mass in the posterior mediastinum, extending to the left of the spine. In these cases as well as in most of the larger herniations a disturbance in the course of the barium mixture is noted during the ingestion of



Figs. 3 and 4, Cases 5 and 6. Small hiatus herniæ, involving cardia of stomach, showing tortuous esophagus and gastric rugæ. Both of these were associated with abdominal malignancy.

the meal. This is not true, however, where the hernia is completely reducible so that there is no abnormal hesitation of the meal at the cardia. When the contents of the hernial sac are adherent above the diaphragm, one may immediately note a torturosity of the lower esophagus, with or without dilatation, and a partial filling of the herniated portion of the stomach.

In the presence of the smaller and of all easily reducible herniæ, there is no abnormality noticeable during the ingestion of the meal. These will, therefore, usually be overlooked by those who fail to examine their patients in the horizontal positions. Several of our cases were partially demonstrated in the vertical position, during the manipulation of the stomach, by forcing the barium mixture upward through the hiatus. The best and most complete demonstration of the hernia can be made in the supine position, while the horizontal oblique positions help to separate the shadows of the esophagus and stomach, and to demonstrate the tortuosity of the esophagus with its termination at the cardia. Occasionally the Trendelenburg position is required to fill the hernial sac. Åkerlund, who has demonstrated a number of smaller herniations, has used a position midway between the supine and the right lateral position. In this position the cardia is the most dependent portion of the stomach, and can thus be completely filled with the barium mixture. Plates are best made in the supine or Trendelenburg position.

To distinguish the type of hernia present it is necessary to establish the relationship of the lower end of the esophagus to the herniated portion of the stomach. This can be readily done in most cases.

One might summarize the roentgenologic findings as follows:

1. Demonstration of a protrusion of a portion of the stomach, with or without the subphrenic portion of the esophagus, through the esophageal hiatus. This may be demonstrated either by air distension, or by filling with a barium suspension, or by a combination of these two methods.

2. Distortion of the lower portion of the esophagus. This may consist of tortuosity with mild dilatation, or of pressure from the herniated mass.

3. Demonstration of the dilatation of the hia-

tus, marked by a wide communication between the stomach and the herniated pouch, with the rugæ of the gastric mucosa extending into the herniated portion.

4. Reduction in the size of the cardiac portion of the stomach, or changes in the contour of the stomach. The gas bubble of the subphrenic portion may be reduced in size, or may be totally absent, and this portion of the stomach is usually displaced medially.

In the differential diagnosis we have to consider eventration of the left dome of the diaphragm, esophageal diverticulum, para-esophageal diverticulum of the diaphragm, diverticulum of the cardia of the stomach, and relaxation of the cardiac orifice of the stomach with dilatation of the subphrenic portion of the esophagus. Occasionally a marked elevation of the diaphragm, producing a tortuosity and dilatation of the esophagus, will present a picture which may be confused with hiatus hernia.

The differentiation can be made in most cases by careful fluoroscopic examination. In eventration of the left dome of the diaphragm one notes an abnormal elevation, but a maintenance of the rounded contour. There is either a greatly diminished response to respiration, a paradoxical excursion, or no excursion at all. The entire stomach will be found under the diaphragm, with upward displacement corresponding to the degree of elevation. The esophagus may be tortuous, but can be traced through the hiatus into the stomach. The stomach will be found in close association with the splenic flexure of the colon which may be demonstrated with barium or may be identified by its gaseous content. The heart is usually displaced to the right.

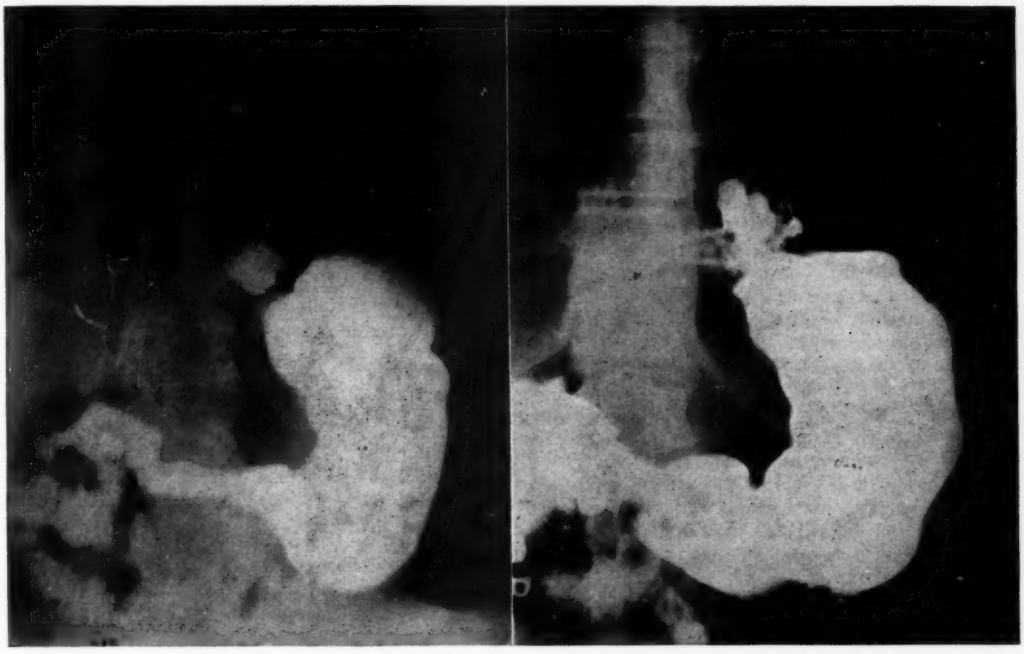
Diverticulum of the lower end of the esophagus or of the adjacent portion of the cardia of the stomach may be differentiated by its subphrenic position, by demonstrating its narrow opening into the esophagus or stomach, and by its rounded contour. It is usually distended with gas, and a fluid level can be demonstrated in the erect position. When such a diverticulum is lodged in a corresponding para-esophageal diverticulum of the diaphragm, as in the cases described by Andrews, Blaker, and Quenus, the roentgenologic differentiation from hiatus hernia is practically impossible. Diverticulum of that portion of the esophagus lying immediately above the diaphragm is comparatively rare, but constitutes a most difficult problem in differential diagnosis. Examination in various positions, complete filling of the sac, and identification of the structures in question should result in a correct conclusion.

With atonicity and relaxation of the cardiac orifice of the stomach, we frequently find a dilatation of the lower esophagus with regurgitation, and this may be most difficult to distinguish from hiatus hernia. The differentiation may be even more difficult where it is associated with elevation of the diaphragm produced by increased intra-abdominal pressure. This may produce a considerable tortuosity of the lower third of the esophagus and thereby present a picture which may closely simulate the smaller herniæ. It should be possible, however, to demonstrate the absence of an actual sac, the subphrenic position of the cardiac orifice, as well as the lack of the characteristic distortion of the stomach.

The symptoms produced by hiatus hernia vary with the type and with the size of the hernial sac, and with its contents. The smaller herniæ are usually not the source of any marked distress. The larger herniæ may produce substernal or epigastric pain. This is the most common symptom mentioned in the literature. Åkerlund found it in sixteen of his cases, but a number of these had other findings which might account for the pain. In our series, eleven complained of epigastric pain or distress, with other pathologic conditions to account for the pain in four cases. One of our cases, in which an obstruction of the pylorus was produced by herniation, suffered periodic attacks of severe pain accompanied by vomiting. Another case experienced distress only when lying on the back or right side. Examination of this patient revealed herniation in these positions, but complete reduction of the hernia in the erect position.

Nausea and vomiting occurred in six of our cases, and is frequently reported in the literature. Belching was also present in six cases. Dysphagia occurred in three, and pyrexia in two cases. Alkalies gave relief from the pyrexia.

The treatment of hiatus hernia is predominantly surgical. The abdominal approach is more advantageous, especially where the content of the hernial sac is readily liberated. The hernial sac is obliterated, if possible, or the communication between the peritoneal cavity and the sac is closed, with a repair of the hiatus. If the con-



Figs. 5 and 6, Case 11. Fig. 5 shows hernia before manipulation. Fig. 6 shows same case after manipulation, demonstrating variation in size of the hernial sac. This figure also demonstrates the gastric rugæ extending into the herniated portion of the stomach.

tent of the hernia is adherent, the transpleural route is indicated. Three of our cases submitted to surgical repair. One of these (Case 1) died of peritonitis. The second case (Case 12) survived the operation, but without relief from symptoms. Subsequent roentgenologic examination revealed repair of the hernia but with still some dilatation of the lower esophagus. Still later examination revealed a recurrence of the hernia. The third case (Case 11) made an uneventful recovery. Later findings in this patient are described under detailed case report. A phrenicotomy was performed in another case. There was no apparent immediate relief. Some months later the patient was put to bed for several weeks, which resulted in a marked improvement in her condition.

Medical treatment consists of dietary measures and of attempts to avoid exacerbation of the condition. Mild laxatives may be indicated to avoid abnormal exertion during defecation. Other forms of excessive exertion must be avoided. Soft foods are indicated to avoid irritation of the lower esophagus and of the cardia, and this is especially true where dysphagia is a prominent symptom. Rest in bed with a liquid diet may be necessary temporarily if dysphagia becomes marked. If the symptoms are associated with certain positions, postural treatment is indicated.

We are reporting herewith nineteen cases of hernia of the esophageal hiatus. In this series there are fifteen females and four males. The age incidence is from 40 to 75, with an average of 58.

In reporting these cases, the authors acknowledge the courtesy and assistance of Drs. H. L. Ulrich, J. D. Lyon, Macnider Wetherby, M. H. Nathanson, and Kenneth Phelps.

Case 1.—B. S., female, age 45, widow. The patient was admitted to the Minneapolis General Hospital September 25, 1928, with the complaint of pain in the epigastrium and vomiting. Her present illness began September 20, 1928, when, shortly after a meal, she was seized with a severe pain in the epigastrium which was soon followed by vomiting. The pain was sharp and shooting in character with a constant feeling of heaviness. Emesis gave temporary relief. She expelled all food taken subsequently, and all food intake brought on an attack of pain.

The patient stated that she had had an attack similar

to the present one some twenty-one years ago, four days after her first childbirth. That attack lasted only a few hours. On the eleventh day after that childbirth she had had a second attack which was also readily relieved. There had been no further attacks until about a year ago, when she had several mild attacks of a similar nature. She had gone through three subsequent pregnancies without any trouble.

The patient was very obese. There was some tenderness in both upper quadrants and in the epigastrium. The physical examination was otherwise negative.

Clinical impression: Chronic cholecystitis.

Roentgenologic examination (Oct. 3, 1928): Fluoroscopic examination of the chest reveals a rounded area of translucency at the left lung base and posterior to the cardiac shadow. This is about the size of a large orange. The lower esophagus is markedly displaced toward the right and lies in close association with this mass. It passes through the hiatus and its subphrenic portion terminates in normal position at the cardiac orifice of the stomach. The cardiac portion of the stomach is in normal position and is not reduced in size. The stomach is somewhat dilated and has the appearance of a large rounded ball. The antrum of the stomach points upward toward the diaphragm. The pylorus and duodenum cannot be demonstrated in the erect position. In the supine position the area of translucency in the posterior mediastinum becomes filled with barium. The antrum of the stomach is pulled up into this region. The barium passes in very small quantities into the duodenum, which extends into the region of the hiatus. The duodenal bulb cannot be demonstrated. The entire study indicates a herniation of the antrum, pylorus and duodenal bulb through the esophageal hiatus. In the erect position the herniated portion of the stomach does not remain filled, but the hernia is not reduced. At the six hour examination there is still about 90 per cent of the barium retained in the stomach, and eleven days later there is still about a 30 per cent retention.

Conclusion: Hernia of the gastric antrum, pylorus and duodenal bulb through the esophageal hiatus, with subtotal obstruction (Figs. 1 and 7).

On a liquid diet the patient ceased vomiting and the liquids apparently passed the pylorus although the heavier barium passed only very slowly. The patient refused surgery and was discharged from the hospital on Oct. 20, 1928.

On Feb. 6, 1929, she returned to the *x*-ray department for re-examination. At this time the entire cardiac portion of the stomach, including the lower end of the esophagus, was herniated through the hiatus, while the antrum and pylorus were in normal position. A distinct change had therefore occurred and the Type II hernia had become a Type III. Further study on Feb. 18, 1929, revealed a recurrence of the herniation of the antrum through the hiatus, with the cardia again in normal position.

The patient submitted to surgery on Feb. 26, 1929. A marked dilatation of the hiatus was found. This was large enough to admit the surgeon's entire hand. The entire stomach was herniated through this opening. The operative procedure consisted in replacement of the stomach into the abdominal cavity, with repair of the hernial opening, and fixation of the stomach against the abdominal wall. The gallbladder was found full of stones, and was removed. During its removal it ruptured with some contamination of the peritoneal cavity. The patient developed peritonitis, and died about two weeks postoperatively. A postmortem examination confirmed the previous findings.

This case is the only one in our series of Type II of Åkerlund's classification, although at one examination it temporarily changed into Type III. All of our other cases are of his Type III.

Case 2.—B. R., female, age 65, married, four pregnancies. The patient was first seen by Dr. M. H. Nathanson in December, 1927. For several years she had suffered considerable pain and the sensation of pressure in the epigastrium. This was intermittent in character. She also complained of a choking sensation associated with the epigastric pain.

The patient was short in stature and moderately obese. The physical findings were essentially negative. Roentgenologic examination by Dr. Nathanson in December, 1927, revealed the abnormality of the cardia, but because of the persistence of the symptoms she was sent to us for further confirmation of the diagnosis.

Roentgenologic examination (April 12, 1928): The esophagus is markedly tortuous in its lower third, and opens into a portion of the stomach which is herniated through the esophageal hiatus. This is about the size of a large orange. It shows a considerable amount of gas and a fluid level within it, and does not empty completely even in the erect position. It connects with the subphrenic loculus by a rather narrow channel. The subphrenic loculus is considerably distorted with a comparative reduction in the size of the cardiac portion and a displacement medially by the tension toward the hiatus. The stomach is otherwise normal.

Conclusion: Hernia of the cardia of the stomach through the esophageal hiatus.

The patient was next referred to surgery for treatment. A phrenicotomy was done in order to relax the left dome of the diaphragm. This gave no apparent relief. In July, 1928, she was placed on bed rest for several weeks, during which time the symptoms improved markedly. Dietary measures were also instituted. The patient is now relieved of practically all of her previous symptoms.

This case illustrates a type in which there is a rather large hernial pouch which produces marked symptoms. Cases 3 and 4 are of a similar nature and will not be recorded in detail. Case 3 demonstrated a fairly large reducible hernia which produced symptoms only in the horizontal position.

Case 5.—C. O., female, age 69, married. The patient was admitted to the Minneapolis General Hospital on

May 24, 1927. She complained of pain in the left upper abdomen, abdominal distension, nausea and vomiting. These symptoms had been of four weeks duration, and had been preceded by indefinite malaise, with some pyrosis. Emesis had been persistent since its onset. There was no weight loss. Diarrhea had been present for two days before admission.

The past history was essentially negative except for a fall down a stairway at the age of twenty, resulting in fracture of ribs. There had been one pregnancy.

The predominant physical finding was a marked ascites. This was relieved by repeated paracentesis. The fluid was found to be a transudate which contained numerous small cystic bodies measuring 8 to 9 mm. in diameter. Microscopically these showed an epithelial lining with loose connective tissue stroma, suggestive of cystic carcinoma of the ovary.

Roentgenologic examination (May 25, 1927): The esophagus is moderately dilated and markedly tortuous in its lower third. There is protrusion of the cardiac end of the stomach through the esophageal hiatus into the posterior mediastinum. The terminal end of the esophagus lies about two inches above the diaphragm. The herniated portion of the stomach is about the size of a hen's egg. It is somewhat angular in outline, and communicates freely with the subphrenic loculus through a greatly dilated hiatus. The gastric rugæ can be clearly traced into the upper loculus. The portion of the cardia below the diaphragm shows a marked diminution in size as compared to the normal, with considerable displacement toward the midline. The hernial sac remains fixed in the posterior mediastinum indicating that it is adherent.

Conclusion: Hernia of the cardia of the stomach through the esophageal hiatus (Figs. 4 and 7).

An exploratory laparotomy revealed extensive carcinomatosis of the peritoneal cavity. The left fallopian tube was thought to be the site of a primary adenocarcinoma. The patient died on October 19, 1927. Autopsy examination was made by Dr. C. J. Watson. The extensive carcinomatosis was again demonstrated. The esophageal hiatus was studied. It was found to be about twice the normal in size, with herniation through it of the lower end of the esophagus and of a loculus of the stomach. These were adherent in the posterior mediastinum and could not be replaced into the abdominal cavity. The layers of the hernial sac were not studied microscopically.

This case is associated with increased intra-abdominal pressure, which probably bears a definite etiologic relationship to some forms of hiatus hernia. Cases 6, 7, 8, 9 and 10 are of similar nature and are not described in detail. All of these five cases died from the effects of intraperitoneal malignancy.

Case 11.—T. O., female, age 56, married, eight pregnancies. The patient was admitted to the Minneapolis General Hospital on March 26, 1929, complaining of lower abdominal pain, with recent nausea and vomiting. She got relief from belching and from lying on her right side. She had qualitative and quantitative food distress. She was markedly constipated. An inguinal hernia had been repaired six months previously.

Physical examination revealed lower abdominal tenderness which seemed to be diffuse. There was a mild secondary anemia.

Roentgenologic examination (March 29, 1929):

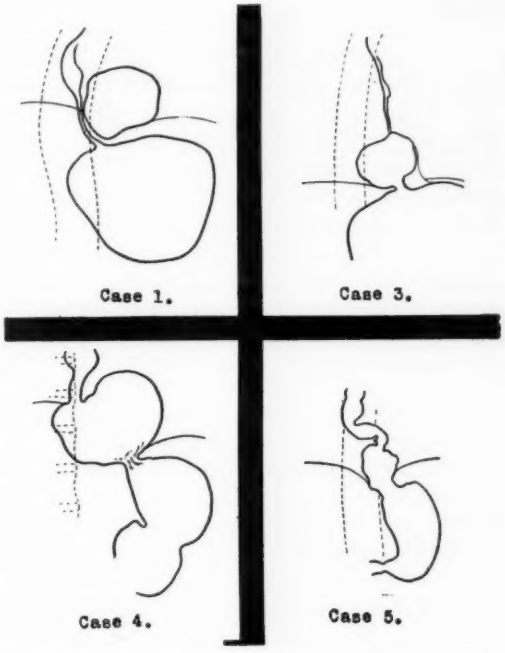


Fig. 7. Diagrammatic tracings of four cases of hiatus hernia. Cases 1, 4 and 5 are also illustrated in Figures 1, 2, and 4, respectively.

There is a relaxation of the esophageal hiatus with herniation of the cardia of the stomach through the hiatus into the posterior mediastinum. The lower esophagus is tortuous and somewhat dilated. The hernia is reducible in the erect position. In the prone position it at first is about the size of a walnut, but on manipulation can be enlarged to the size of a small orange. The gastric rugæ are demonstrated extending into the herniated sac. The subphrenic portion of the stomach is displaced medially. The stomach is otherwise normal. The colon shows multiple diverticuli throughout its extent. Examination of the gallbladder by the Graham-Cole method demonstrates normal function and evidence of pericholecystic adhesions.

Conclusion: 1. Hernia of the cardia of the stomach through the esophageal hiatus. 2. Diverticulosis of colon. 3. Pericholecystic adhesions (Figs. 5 and 6).

The patient submitted to surgery. The multiple diverticuli of the colon and the pericholecystic adhesions were demonstrated. The esophageal hiatus was found to be dilated to a diameter of about one inch with a hernial pouch extending through it into the posterior

mediastinum. The opening was repaired and the cardia of the stomach was fixed to the parietal peritoneum. The postoperative course was uneventful.

Later examination revealed a small traction diverticulum of the lower portion of the esophagus just above the diaphragm. The cardiac portion of the stomach was now in normal position, but showed an hourglass constriction. This was interpreted to represent the site of the previous constriction of this portion of the stomach at the hiatus.

This case illustrates a smaller type of hernia which is usually associated with rather indefinite epigastric distress. In these cases the hernia is frequently reducible, and is usually not demonstrable in the erect position. Filling of the hernial sac is produced in the supine or prone position. The presence of inguinal hernia and of colonic diverticuli adds interest to this case, and points toward the congenital origin of these herniæ. Cases 12, 13, 14, 15, 16, 17, 18 and 19 are very similar to the above described case, and the details of these cases are therefore omitted.

SUMMARY

1. A group of nineteen cases of hernia of the diaphragm through the esophageal hiatus is reported, and the detailed clinical and roentgenologic findings in four representative cases are given.

2. Hiatus hernia has been considered an infrequent condition until recently. Our own experience substantiates that of Healy, Åkerlund, and others, that this is a comparatively common condition, and that it is far more frequent in occurrence than all other types of diaphragmatic hernia.

3. Hiatus hernia is rarely diagnosed clinically. The symptoms are usually vague, and often point toward other types of pathology.

4. Roentgenologic examination is the usual method of establishing this diagnosis. A careful fluoroscopic study with the aid of an opaque meal must be made. The patient must be examined in various positions, as use of the vertical position only will frequently result in overlooking this condition.

5. Hiatus hernia must be differentiated from (a) eventration of the left dome of the diaphragm, (b) esophageal diverticulum, (c) paraesophageal diverticulum of the diaphragm, (d) diverticulum of the cardia of the stomach, (e) relaxation of the cardiac orifice of the stomach with dilatation of the subphrenic portion of the esophagus, and (f) tortuosity and dilatation of the esophagus produced by elevation of the diaphragm due to increased intra-abdominal pressure. The differentiation rests entirely on the roentgenologic observations.

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THE AFFERENT NERVE SUPPLY OF BLOOD VESSELS*

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SINCE the introduction of dissection as a means of studying the mechanisms of the body, it has been common knowledge that the blood vessels are richly supplied with nerves. Their function, however, was totally unknown—one might even say unsuspected—until Claude Bernard[1] in 1852 coincidently with Brown-Sequard[2] made his startling discovery that section of the sympathetic nerve in the neck of the rabbit caused dilatation of the vessels in the corresponding ear. This demonstration of a nervous control of blood vessels started a vast amount of research upon this interesting and very important subject, research which has resulted in an immense literature and a definite knowledge of the motor control of blood vessels. However important the vasomotor system may be, it is only one-half of the reflex arc which causes the reaction to appear. Although a large amount of work has been done upon vascular reflexes arising from extra-vascular sources, yet the afferent paths from the vessels themselves have been studied very little.

Probably the first important work upon this subject was done by Heger[3] in 1887. Working upon the vessels of the hind limb, he discovered that excitation of the internal surface of the blood vessels can cause a general change in the circulatory conditions at a distance. He stated that it was a reflex phenomenon set up by the stimulation of sensory nerve endings in the vessels themselves. Chemical stimulation of the vascular endothelium in his experiments gave most often a rise of general blood pressure accompanied by a slowing of the heart, and only exceptionally did he get a lowering of the blood pressure. This work was not repeated until 1896, when Spallitta and Consiglio[4] confirmed his results, and added the very interesting information that not only chemical stimuli but also "simple oscillations" of the blood pressure could put this reflex pathway into activity. These results were in turn confirmed by Delezenne.[5] Pagano[6] in 1900 found that the carotid and vertebral arteries respond with general blood pressure and respiratory changes to injections into them of prussic acid and nicotine. The Swedish surgeon, Lennander,[7] studied local anesthesia and the sensitivity of organs in 1901. In 1922 Odermatt[8] in a lengthy paper discussed this problem and came to the conclusion that irritating chemical substances injected into the blood stream cause blood pressure changes and pain only when they reach the capillaries, while the arteries and veins do not respond to such stimuli, but do respond to stretching of their walls or manipulation in the same way. He further adds that this sensation from the arteries and veins is due to the periarterial plexus of nerves in the adventitia, and is lost upon destroying these nerves (as by periarterial sympathectomy). Ranson, Windle, and Faubion[9] in 1923 assumed a reflex excitation of the respiratory center through sensory nerve endings in the vessel walls to explain the immediate increase of respiratory rate after the intra-arterial injection of ether. Freidrich,[10] in reporting a case in which pain in the hand was relieved by periarterial sympathectomy after section of the three arm nerves had failed to cure it, said that in dogs under partial anesthesia injection of 50 per cent lactic acid into the femoral artery caused pain, but not if the artery was ligatured above the site of injection. He concluded that there are nervous paths in the arterial walls which conduct impulses producing pain.

In opposition to the ideas presented by previous authors, Dennig,[11] in 1924, found no sensory nerves in the adventitia of the femoral arteries in dogs. In 1925 the same author[12] claimed that there are no indications of long sensory nerves in the adventitia of arteries, and furthermore that lactic acid causes pain if the adventitia is removed without ligation of the artery.

This observation lends weight to the most evident criticism of all the previous experiments. Those experiments were carried on in the intact animal, and the injection was made into arteries that were not isolated from the general circulation. Thus the possibility of direct stimulation

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of the vasomotor center was excluded only by the assumption that the time that elapsed before the appearance of symptoms (10-12 seconds) was too short for the circulation to have carried the injected substance that distance. As a matter of fact, the time required for blood to circulate through the body is very short, and the exact time necessary for the blood to flow from one certain part to another cannot be predicted with any degree of accuracy.

However, the general trend of opinion seems to be that there are sensory nerves in the walls of arteries and veins and around capillaries which respond to stimulation with pain and blood pressure changes.

This conclusion is borne out by clinical experience. Operations performed under local anesthesia point in some cases to sensory endings in the blood vessels responding to manipulation with the sensation of pain. Bier,[13] before Lennander, showed that ligation of the mesenteric arteries is painful. Hotz[14] said that an increased novocaine concentration caused pain in arterial anesthesia. Ligation of arteries often causes pain. In insufficiently anesthetized patients in goiter operations, ligation of the superior thyroid artery often causes pain, while ligation of the inferior thyroid never causes pain. Ligation of the four main arteries of the stomach is painful under local anesthesia. In addition to the above, Odermatt states that pain is caused by arterial and intravenous injections, injury to vessels and traumatic aneuryism, thrombi, emboli, arteriosclerosis and other vessel diseases, and by vessel cramp. Hess and Konigstein[15] say that changes in the fullness of the vessel are frequently accompanied by pain.

Clinical relief of pain in various arterial diseases by periarterial sympathectomy, first introduced by Leriche, also points to pain conduction by the arteries. Cain and Hamburger,[16] Freidrich,[10] Leriche,[17] G. E. Brown,[18] and many others have reported relief of pain in Raynaud's disease, thrombo-angiitis obliterans, and related vascular affections, after periarterial sympathectomy or after lumbar sympathetic ganglionectomy in conjunction with that operation. In one case, the severe and uncontrollable pain of angina pectoris was immediately stopped by cervical sympathectomy. These results might be taken to point toward the conduction of pain along the arteries, but they are also amenable to another explanation, that is, that the pain is caused by the anemia of the parts affected, and the relief was gained, not by sectioning the sensory nerves carrying the impulses, but by the increased vascularity of the region after the operation. In the main, however, clinical experience may be said to uphold the idea that sensory nerves are present in vessels, at least at certain places. These might give rise to vascular reflexes.

An examination of the blood vessels histologically in respect to their nerve supply is very illuminating. It has long been known that the arteries are well supplied with nerves. The existence of vasoconstrictor nerves in all organs of the body except the lungs, brain, heart and voluntary muscle has been proved beyond any shadow of a doubt (Bayliss[19] 1923). Bradford and Dean[20] in 1894 showed that the lungs were supplied with vasomotor nerves. Some doubt was cast upon the existence of vasomotor nerves in the lungs by later work, especially that of Brodie and Dixon[21] in 1904. However, Fuhner and Starling,[22] in 1913, settled the question when they showed that adrenalin constricts the branches of the pulmonary artery. The cerebral circulation was found by Bayliss and Hill[23] to be controlled only by the general arterial and venous pressures. Later workers, by staining methods, have shown the presence of nerve fibers in the blood vessels and pia of the brain (Obersteiner[24] 1897, Morison[25] 1898, Huber[26] 1899, Rohnstein[27] 1900, Hunter[28] 1901). However, they had no definite means of deciding upon their function. They may be either sensory or motor. It seems very unlikely that the heart is supplied with vasomotor fibers (Markwalder and Starling[29] 1913). Evidence has been brought forth to show the existence of vasoconstrictor fibers in striated muscle by Gaskell.[30] His experiments were confirmed by Hartman, Blatz and Kilborn.[31] But the evidence is not conclusive enough to say that the question is decided (Bayliss[19] 1923).

Since the introduction by Leriche[17] of his periarterial sympathectomy in which he removes the adventitia of arteries in order to relieve vasomotor diseases, there has been done a good deal of work to explain the varied results of this operation. Nerve plexuses have long been known to exist around arteries and veins. Berglas,[32] with a binocular microscope, "was able to dissect the periarterial plexus around such vessels as the iliacs, where he always found large nerve strands at the union of the adventitial and mus-

cular coats." He believes that this plexus extends along the whole vascular tree and that it is everywhere continuous. Periarterial sympathectomy would effectively remove this plexus. However, physiological experimenters, especially Langley,[33] Wiedhoff,[34] and Schlif,[35] have found that in animals the normal vasomotor reactions are not destroyed by this operation, while, if the main nerves to the limb are cut, those reactions are lost. Woollard[36] in some very careful histological work explains these results very satisfactorily. He studied whole preparations of such small animals as the rat and guinea-pig and was thus able to "examine the innervation of the vessels in unbroken continuity." In addition he removed the abdominal sympathetic chain on one side in some cats and allowed the nerves to degenerate; while in other cats he removed the adventitia from the common and external iliac arteries on one side and allowed these nerves to degenerate. The conclusions to which his experiments led him are very interesting. He found that the "limb vessels are innervated proximally by nerves derived directly from the aortic plexus, and distally by contributions from the adjacent nerve trunks." That is, he found that above the bifurcation of the femoral artery the nerve supply consists almost entirely of non-medullated nerve fibers (considered not to be sensory) derived from the sympathetic plexus around the aorta. Distal to the bifurcation of the femoral a second mode of innervation adds itself to the first. It consists in many nerve fibers, usually too small to be seen grossly, which run into the periarterial plexus and fuse with it. These fibers are largely medullated (at least four to one). Thus part of the distal innervation of the arteries is derived from the ordinary somatic nerves, and these nerves may be sensory. He further describes medullated nerves which send branches both to the blood vessel wall and to the surrounding fat and connective tissue. These may form a neural basis for the axon reflex. In the capillaries, however, he did not find a complete innervation. Sympathetic fibers were found at various parts, but there was no evidence of innervation by medullated nerves. However other authors have reported the presence of abundant nerve fibers around the capillaries. Dogiel[37] has described terminal nerve plexuses around capillaries and small blood vessels. Yet it is not certain whether his work was really upon capillaries or merely upon small arterioles. Shafer,[38] however, states that "gold impregnation of the rabbit's mesentery shows every capillary to be supplied with a nerve running along it, the nerves forming loops." Thus there is found to be a very definite histologic basis for vascular reflexes, both the sensory and motor pathways being potentially present.

Vascular reflexes arising from the smaller vessels are still problematical, but other types of reflexes affecting the blood vessels are well known. Vascular reflexes may be divided into two classes, those with the sensory stimulus arising from extravascular structures, and those with the sensory stimulus arising from the vessel itself. The most important examples of the first type are the following. It is well known that stimulation of the central end of any cutaneous nerve will cause an immediate rise of blood pressure under most circumstances. Thus it is evident that cutaneous stimulation or in fact any stimulus that would cause pain will affect the vasomotor center as well as the respiratory center. Pressor responses may also be elicited by stimulation of visceral nerves, or by an insufficient blood supply to the brain. The axon reflex of Langley may be mentioned here although it is not a true reflex. It has been suggested by Bayliss[39] that this reflex is brought about by stimulation of a sensory nerve which causes dilatation of the blood vessels of the region by a peripheral nervous mechanism. This has been shown to be accomplished by an axon which has two collateral terminations, one going to the skin and the other to the blood vessel. Ninian Bruce[40] brought forth experimental evidence in support of this view. He found that the application of oil of mustard to the skin caused a vasodilatation even if the nerve had been recently cut, and therefore the effect could not be a spinal one as Spiess[41] had thought. These observations have been borne out by histological studies.

There are only two definitely proved examples of the second type. They are the depressor reflex and the carotid sinus reflex. The depressor nerve was first discovered by Cyon and Ludwig[42] in 1866. In the rabbit it is a small nerve arising from the base of the aorta, as shown by Koster and Tschermack,[43] and running near the vagus but separate from it. In other animals it is probably incorporated in the vagus nerve. If this nerve is cut there is no response upon stimulating the peripheral end, but stimulation of the central end results in an immediate and very

marked fall in the blood pressure. This fall has been shown by Bayliss[44] to be due mainly to dilatation of the splanchnic vessels, but also, in part, due to a general vasodilatation throughout the entire body. The normal stimuli for this reflex are supposed to be blood pressure changes in the aorta. Therefore this constitutes a true vasovascular reflex. The carotid sinus reflex was first demonstrated by Sollmann and Brown[45] in 1912, who found that traction upon the cephalic end of the carotid artery produced a reflex fall of blood pressure. They found the effect to be due almost entirely to a slowing of the heart. Vasodilatation plays little or no part. At the same time they were unable to locate either the afferent or the efferent path, for section of the vagus nerve did not interfere materially with the reflex. Hering,[46] however, in 1924 traced this reflex to a branch of the glossopharyngeal nerve which he calls the sinus nerve. He says that stimulation of the central end of this nerve causes a greater lowering of the blood pressure than does the stimulation of the depressor nerve. These are, then, true vascular reflexes such as we have been discussing. Their function is evidently that of controlling the blood pressure to prevent damage to the vascular system in case the pressure should become too high, as well as to regulate the normal blood pressure. On considering other protective mechanisms in the body, it becomes evident that they are reduplicated time and again in various places so that there may be as little likelihood as possible that the body will be caught unprepared. Especially in the blood vascular system, the single system most essential to life, would one expect such precautions. Another very likely function for such a vascular reflex mechanism would be the regulation of blood pressure in the various parts and the coördination of general blood pressure to their needs.

Briefly summarizing the present status of this question of sensory nerves from blood vessels, one would say that the evidence points toward the presence of sensory fibers from the capillaries which respond to chemical substances in the blood-stream, and the presence of sensory fibers from the larger vessels which respond to pressure changes and manipulation. Histologically this idea is borne out except that the largest vessels, that is, above the bifurcation of the femorals, may have no sensory fibers. In addition, microscopic study shows that the paths of these fibers are largely through the peripheral nerves. This conception, however, has never been proved conclusively. The experimental work is very difficult to interpret. Since this is a very important physiological mechanism, it seemed advisable to investigate the possibility of such reflex pathways experimentally.

EXPERIMENTAL WORK

A series of preliminary experiments was carried out along the line of investigation used by the older experimenters, that is, by the use of irritating substances injected into the arteries and veins. This method of procedure was found to be unsatisfactory because of the inconstancy of the results, and because of the multitude of uncontrollable factors that made the results unreliable and their interpretation difficult. Therefore it was decided to conduct experiments using pressure changes within the vessel as the stimulus.

To this end the following apparatus was evolved. A mercury manometer was connected with a three-way tube. From one arm of this tube a length of rubber tubing led to an ordinary automobile tire valve arranged so that an automobile tire pump could be connected to it. From the other arm a tube was connected to a metal tube 1.5 to 3 millimeters in diameter. This was soldered at one end, but had an opening two inches from that end. Near the tip of this metal tube a circular indentation was made and about four inches from this another groove was made around the tube. A piece of fine tissue drainage tubing was then stretched over this portion of the tube and was tied with thread at the groove, making a long balloon. Thus, a stroke of the pump would distend the balloon and at the same time cause the mercury in the manometer to rise and measure the pressure. The manometer was three feet tall, so to write the pressure upon the revolving drum, a volume recorder was connected to the manometer, and the pointer of the volume recorder was adjusted to write upon the drum. The scale of reduction was then determined by actual measurement at different pressures. The balloon was inserted into the artery or vein that it was desired to study.

With this apparatus it was possible to have a definite measured pressure as high as seven hundred millimeters of mercury in the artery, and at the same time have this pressure written upon

the drum. The balloon did not burst under these pressures because the artery or vein acted as an outer casing exactly like an automobile tire. Several of these balloons could be connected to the same pump to study the effects in various places.

EXPERIMENTAL RESULTS

These experiments were carried out upon dogs. It was found to be necessary to use dogs weighing thirty-five pounds or more so that the arteries would be large enough to receive the balloon easily. The experiments were carried on without the use of morphine. The only substance used which would tend to deaden the reflexes was the ether which was used for the anesthetic. In one dog this was dispensed with by decerebrating and allowing time for the ether to be eliminated from the body (two and one-half hours). Indeed, two of the dogs were made hyperirritable by the injection of 3 cubic centimeters of a four-one hundredths per cent solution of strychnine sulphate. This procedure caused such a tremendous hyperirritability that merely touching a finger to the skin over the sternum would cause a reflex rise of blood pressure. This injection of strychnine was given to eliminate the least trace of suspicion that the dogs were in poor condition and that the uniformly negative results might be due to a lowered irritability of the animal rather than to an absence of nerve endings, which would respond with reflex blood pressure changes to distention of the vessel walls. In the other dogs it was considered unnecessary to inject strychnine because, in those dogs, the carotid artery was used, which gave a marked fall of blood pressure upon distention. This was believed to be sufficient indication that the reflexes were working properly. However, in all these dogs, the irritability was controlled very carefully by stimulation of the femoral or sciatic nerves electrically. This was done to afford a means of comparison between the reactivities of the various dogs as well as to prove that the normal blood pressure reflexes were in good condition. This stimulation was accomplished with an ordinary induction coil with the secondary coil set at eight centimeters. The source of electricity consisted of two dry cells. The nerve was stimulated by means of a Sherrington electrode in the case of the sciatic nerve and by merely laying the nerve upon electrodes with the femoral nerve. The nerves were stimulated at intervals throughout the experiments. Stimulation usually caused an immediate rise of blood pressure followed by a drop to below normal, after which recovery to normal occurred. The relative irritability as shown by nerve stimulation is brought out clearly by the accompanying table.

TABLE I

	NERVE STIMULATION		
Dog No.	Nerve	Av. Rise of Blood Pressure	Av. Fall of Blood Pressure
1	Femoral	12.3 mm. Hg.	7.7 mm. Hg.
2	Sciatic	15.	15.
3	Sciatic	16.6	24.2
4	Femoral	6.	13.

Table I shows that the dogs were irritable.

The balloons were inserted into the vessels of these dogs in much the same manner as cannulæ. The vessel was cleaned and ligatured away from the site of insertion. A clamp was then placed on the side of the vessel in which it was desired to insert the balloon. A loose ligature was placed around the vessel and the vessel was nicked. The balloon was entered into the vessel, the clamp was removed and the balloon pushed into the desired position. The loose ligature was then tied tightly to prevent loss of blood and to hold the balloon in place. In this way, the balloon was placed ten to twenty centimeters away from the point of operation. Thus the nerve supply was not disturbed in the region of the balloon. The femoral artery or vein was entered in the middle of the thigh, and the axillary artery was entered at the lower border of the axilla.

RESULTS

Probably the best way to show the results of these experiments is to tabulate the effects produced in each vessel by its distention. In each case the second blood pressure measured is at the point of greatest change during the period of stimulation.

It can be seen very easily from the following tables that distention of any of these vessels except the peripheral carotid artery even with extremely high pressures caused absolutely no uniform change in blood pressure. The largest average variation is only one millimeter of mercury.

The femoral artery or vein gave no indication of a reflex change of blood pressure or even of respiration at any time. These vessels were studied carefully from the bifurcation of the aorta to a point just below the knee. In other

TABLE II

PERIPHERAL FEMORAL ARTERY

Balloon Pressure mm. Hg.	Mean Blood Pressure before Stimulation mm. Hg.	Mean Blood Pressure after Stimulation mm. Hg.	Change mm. Hg.
546	134	135	1
630	137	133	—4
672	117	117	0
600	152	156	4
697	146	146	0
663	140	139	—1
680	134	133	—1
714	121	126	5
748	150	152	2
375	152	151	—1
390	153	155	2
435	157	157	0
590	166	164	—2
495	163	159	—4
450	167	161	—6
433	176	180	4
		Total	—1

Average —1/16 = —0.062 mm. Hg.

TABLE III

CENTRAL FEMORAL ARTERY

Balloon Pressure mm. Hg.	Mean Blood Pressure before Stimulation mm. Hg.	Mean Blood Pressure after Stimulation mm. Hg.	Change mm. Hg.
246	77	77	0
226	80	81	1
238	77	79	2
399	153	154	1
672	152	153	1
672	153	153	0
714	151	153	2
756	150	149	—1
		Total	6

Average 6/8 = 0.75 mm. Hg.

TABLE IV

PERIPHERAL FEMORAL VEIN

Balloon Pressure mm. Hg.	Mean Blood Pressure before Stimulation mm. Hg.	Mean Blood Pressure after Stimulation mm. Hg.	Change mm. Hg.
567	143	145	2
604	145	143	—2
525	141	144	3
504	131	133	2
525	135	135	0
		Total	5

Average 5/5 = 1 mm. Hg.

TABLE V

CENTRAL FEMORAL VEIN

Balloon Pressure mm. Hg.	Mean Blood Pressure before Stimulation mm. Hg.	Mean Blood Pressure after Stimulation mm. Hg.	Change mm. Hg.
158	87	86	—1
152	82	86	4
176	92	87	—5
166	87	83	—4
236	87	90	3
220	96	95	—1
222	96	98	2
236	94	93	—1
246	87	90	3
236	94	90	—4
220	77	74	—3
230	72	72	0
240	74	76	2
240	73	73	0
441	150	152	2
630	146	143	—3
600	143	145	2
630	143	143	0
		Total	—4

Average —4/18 = —0.22 mm. Hg.

TABLE VI

CENTRAL AXILLARY ARTERY

Balloon Pressure mm. Hg.	Mean Blood Pressure before Stimulation mm. Hg.	Mean Blood Pressure after Stimulation mm. Hg.	Change mm. Hg.
450	172	179	7
480	187	185	—2
570	196	195	—1
420	185	188	3
555	180	180	0
555	176	173	—3
480	177	176	—1
555	178	176	—2
525	179	181	2
495	176	174	—2
		Total	1

Average 1/10 = 0.1 mm. Hg.

TABLE VII

PERIPHERAL CAROTID ARTERY

Balloon Pressure mm. Hg.	Mean Blood Pressure before Stimulation mm. Hg.	Mean Blood Pressure after Stimulation mm. Hg.	Change mm. Hg.
663	113	94	—19
680	115	103	—12
595	112	99	—13
476	111	102	—9
680	109	88	—21
645	105	86	—19
615	105	86	—19
620	107	80	—27
645	113	96	—17
544	111	90	—21
525	125	116	—9
150	125	122	—3
300	127	116	—11
330	161	142	—19
150	152	147	—5
495	149	127	—22
450	147	131	—16
420	150	136	—14
330	157	145	—12
360	167	156	—11
420	160	152	—8
450	162	153	—9
495	173	158	—15
495	171	149	—22
465	172	138	—34
255	182	154	—28
450	180	151	—29
90	180	180	0
375	178	148	—30
180	178	168	—10
255	179	157	—22
675	177	142	—35
510	179	141	—38
495	129	113	—16
150	126	123	—3
270	132	123	—9
525	131	112	—19
		Total	—626

Average —626/37 = —16.92 mm. Hg.

TABLE VIII

SUMMARY

Vessel	Average Blood Pressure Change in mm. Mercury
Peripheral Femoral Artery	—0.062
Central Femoral Artery	0.750
Peripheral Femoral Vein	1.000
Central Femoral Vein	—0.220
Central Axillary Artery	0.100
Peripheral Carotid Artery	—16.920

words, the iliac, femoral, and popliteal vessels were investigated, and showed no signs of sensory pathways responding to changes in pressure inside the vessels.

The axillary artery and vein were usually rather small and only in one case was it possible to insert the balloon. This was done at the lower border of the axilla so that the balloon covered a stretch of about four inches from the axillary portion of the fore limb to the chest wall and slightly past that. In this portion of the artery four large branches were given off near the central end of the balloon. One branch was as large as the axillary artery itself. No change of blood pressure was noted during the period of distention, but as soon as the pressure was released a slight fall of blood pressure occurred in most cases (averaging two to three millimeters of mercury). This fall of blood pressure was probably due to the opening of these branches again, and was undoubtedly mechanical in nature.

In the carotid artery, however, the case is much different. Here even moderate degrees of distention cause a marked fall of blood pressure which begins five to six seconds after the beginning of stimulation. Furthermore the amount of blood pressure fall seems to be roughly proportional to the amount of pressure in the artery. A still more striking phenomenon is found in the fact that in most cases the blood pressure did not return to normal until the pressure had been released. Doubtless this is a reflex effect. It was found at post-mortem that the tip of the balloon must be almost at the base of the skull to produce any change. When the balloon was 4 centimeters lower down the artery, distention was without effect. Without a doubt this phenomenon is due therefore to the stimulation of the carotid sinus nerve of Hering, which is located at the junction of the external and internal carotid arteries. The balloon was found at post-mortem to lie in this region. The carotid sinus therefore responds to distention of the carotid artery at that point by lowering the blood pressure reflexly.

Since none of the vessels studied except the carotid artery gave any reflex change of blood pressure, and the carotid itself gave a response only when stimulated in a very circumscribed region, it seems probable from these experiments that the larger blood vessels as a whole do not have sensory nerves which respond to changes of pressure within their lumen by reflex changes of blood pressure. It might be that there are other responsive regions like the carotid sinus, but they have never been demonstrated. These experiments, however, have dealt only with increase of pressure within the vessels. The opposite process, decrease of pressure, or collapse of the vessel walls, has not been studied as yet.

It is evident that this mechanism has some function in the maintenance of normal blood pressure. It may also be of importance in abnormal conditions of the vascular system, either of hypertension or of hypotension. The sudden fall of arterial blood pressure due to stimulation of nerves in the region of the carotid artery suggests a possible relationship of this mechanism to the production of some type of reflex shock. Its specific relationship can be only a matter of conjecture at the present time, but further investigation of this subject is warranted along more definitely clinical lines.

CONCLUSIONS

1. Injection methods of studying the sensory nerve supply of blood vessels cannot be depended upon for good results.
2. The carotid sinus can be stimulated by pressure changes in the carotid artery.
3. Pressure changes in the other vessels studied give no reflex blood pressure changes.
4. Therefore it can be assumed that the blood vessels studied (except the carotid artery) have no afferent nerves capable of being stimulated by increased pressure from within, which carry impulses affecting blood pressure.

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BLOOD TRANSFUSION WITH A REPORT OF ONE HUNDRED AND FIFTY CASES*

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IN 1667 Jean Baptiste Denys, physician to Louis XIV, performed a blood transfusion on a youth, introducing sheep's blood into the veins of his patient. Since this courageous piece of work, probably the first successful blood transfusion, much water has flowed under the bridge, or, I suppose one might say, much blood has flowed through the veins. Enjoying at various times, and sometimes at the same time, the enthusiastic reception in certain quarters and bitter opposition in others common to all new ideas, at times heralded as the ultimate panacea for all ills and at others forbidden by royal edict, this procedure has survived the vicissitudes of changing medical thought for two hundred and fifty years. It is only now coming to be regarded as almost entirely devoid of evil consequences and its wide application fairly well understood.

The earliest workers in this branch of surgery were confronted by a host of problems, both biological and technical. Utterly ignorant of blood incompatibility and unable for the most part to overcome the difficulties subsequent to rapid coagulation of their medium, their results were often negative, and almost as often disastrous. The modern use of sodium citrate as an anti-coagulant, and the investigations of Landsteiner, Moss, and Jannsky resulting in the establishment of the four blood groups, followed by the perfection of apparatus for the direct transfusion of blood, have raised this operation to the level where it is no longer viewed as a last desperate resort, but as a therapeutic procedure of almost universal safety and immense benefit in a constantly growing list of ailments.

Of the many conditions benefited by the introduction of fresh blood which are given in the following list, some are obvious and are universally accepted. Several are still open to question and undoubtedly will often fail to give the desired results. There are two or three conditions in which transfusion is still considered dangerous by some workers; however, there is no condition given for which some author has not reported favorable results of transfusions in series of cases. Thus one is led to believe that in even the most doubtful a transfusion is at least worth a trial.

Indications:

1. Acute hemorrhage
2. Chronic hemorrhage
3. Pernicious anemia
4. Severe secondary anemia
5. Hemorrhagic diathesis
   a. Hemophilia
   b. Purpura
   c. Hemorrhagic disease of the newborn
   d. Thrombocytopenia
   e. Melena neonatorum
6. Severe toxemias of
   a. Burns
   b. Erysipelas
   c. Acute intestinal intoxication
   d. Septicemia
   e. Septic pneumonia
   f. Infectious diarrhea
7. Shock
8. Preoperative, for poor surgical risks
9. Postoperative
10. Carbon monoxide poisoning
11. Premature and debilitated infants
12. Starvation in newborn, with dehydration and acidosis
13. Immuno-transfusion in septicemia and bacteriemia

In acute hemorrhage with the loss of a large amount of blood the treatment by means of transfusion is so well established that it hardly merits discussion. That the replacement of the loss in blood volume by a fluid identical with that lost is ideal, is axiomatic, and it is therefore in this type of case that the results are most dramatic. Severe secondary anemias which often require several months treatment to raise the hemoglobin to the desired level and in which the patient is under par during the time the attempt is being made, respond very promptly to the introduction of fresh blood, and moreover in instances of individuals who are apparently unable

*From the Miller Clinic.

to raise their own hemoglobin, the new level left by the transfusion is usually held very well. In pernicious anemia, while blood transfusion is not considered in any sense of the word curative, the injection of whole blood furnishes the best and quickest method of rescuing the patient from immediate danger, and often apparently serves to tide him over into a remission. Morowitz, describing 200 transfusions done for pernicious anemia, goes so far as to state that a large transfusion will always result in a remission, a belief not generally held. He feels that remissions are not caused by a replacement of the blood alone, but by the inhibited destruction of the red cells. Studies in nitrogen metabolism showed a quantity of urobilin excreted in a period of two days following a transfusion for pernicious anemia corresponding to the amount of hemoglobin transfused. None the less, states Morowitz, this same patient shortly had a remission lasting nine months.

In the hemorrhagic diatheses blood transfusion can ordinarily be expected to give relief, although, in view of the work of Lewisohn, Renaud, and others, it becomes a question in certain of these conditions whether to employ unchanged blood or to use a method entailing the use of sodium citrate. In thrombocytopenia with a platelet deficiency and a patient depleted to the point where even a splenectomy constitutes a grave surgical risk, whole blood intravenously is the remedy par excellence.

Cross reports 519 transfusions in children, 122 of which were exsanguination transfusions for the relief of severe toxemias resulting from burns, acute intestinal intoxication, septicemia, toxic pneumonia, infectious diarrhea, etc. Jones feels that no patient whose response to infection is below normal should be allowed to struggle against the infection without the aid of a transfusion. Another author makes a routine practice of performing a preoperative transfusion on all surgical patients whose hemoglobin is below 60 per cent. Moore and Dennis state that in premature infants too weak to assimilate food the increasing of the blood content 50 per cent is a life-saving procedure, and that in starvation of the newborn, with dehydration and acidosis, nothing supersedes blood transfusion.

As to the use of blood transfusion in acute septic states, one is forced to admit that it is in this type of case that the least may be expected, although now and then a result stands out as being little short of a miracle. Immuno-transfusion, using the blood from immunized donors, may prove to be of value in certain cases where there is lacking the normal response of the patient's blood to the infection. Wright demonstrated that in severe septicemia an ordinary transfusion is of little value since as a rule normal blood contains fewer protective substances than the blood of the infected patient. He suggested transfusing with the blood of a donor who had been vaccinated with the patient's infecting organism. Colebrook and Storer describe tests to determine whether a patient should be treated with vaccines or by immuno-transfusion. If it is decided that vaccines would be of no avail, the donor is given a subcutaneous injection of vaccine and when sufficiently immunized his blood is taken and given directly, without the admixture of citrate, to the patient. They report nine cases of immuno-transfusion in which the procedure was of value, and seven in which no benefit was derived. Unger starts the immunization with an initial dose of one billion and gives as much as 100 to 150 billion in a week, the doses being given daily. He states that it requires nine to ten days to develop immune bodies in a donor. He reports forty-two cases of septicemia in which ordinary transfusion was used, of which number nine recovered. In the second group of cases, the donors were vaccinated with organisms from the patients' blood; none recovered. In the third group of fifteen cases, eight received blood from a donor who had been immunized with the organism taken from some other patient; three recovered. Seven were transfused with the blood of a donor who had been immunized with the organism obtained from the patients' blood; five recovered. Our only case of immuno-transfusion was that of a case of subacute bacterial endocarditis, in which there was no apparent benefit.

On the subject of the method to be used in introducing the blood there is much to be said—and probably much that will be ignored concerning each method by the exponents of other methods. The issue at present seems to be the choice of one of two methods, the one employing sodium citrate as an anti-coagulant, and the other utilizing whole, unchanged blood. The citrate method is perhaps simpler and more quickly available for many physicians, the citrate elim-

inating the necessity for hurry, and permitting an operation which hardy admits of total failure. It is, no doubt, quibbling over a trifle to say so, but I feel that whole blood is the ideal substance to be used. In most cases it actually makes very little difference which technic one uses, and with certain exceptions such as, for example, thrombocytopenia with a platelet deficiency, and septicemia where it has been shown that citrate impairs the function of the leukocytes, certainly the technic with which the surgeon is most familiar and does most expertly is the one to use. Lewisohn, in a study of citrate transfusions after ten years, believes this method to be the simplest and says that reactions are not due to citrate, also that direct methods prevent the use of a cannula in the median cephalic vein in infants. I can only say from personal experience that our reactions were materially reduced when the change was made from the citrate to the direct method, and also that I have never failed in an attempt to get a cannula into the veins of an infant.

Rosenthal and Baehr have shown that there is a marked shortening in the coagulation time following the intravenous injection of sodium citrate. This becomes maximum in one hour and persists for many hours. The action of sodium citrate in vivo is directly opposite to that in vitro, the platelets in the circulating blood being destroyed with the gradual liberation of thromboplastic substances. The number of platelets begins to decrease immediately and the maximum reaction is obtained in fifteen minutes with a return to normal in one hour. No change is noted in other factors such as calcium, fibrinogen, and anti-thrombin. The shortening in the clotting time fails to occur in individuals with a diminished number of platelets. Thus there is every reason to believe that there is a definite contra-indication to the use of citrated blood in purpura hemorrhagica or thrombocytopenia where there is a dangerous decrease in the number of platelets.

On the other hand Tzavaru, Mavrodin, Renaud, and others report series of cases of hemorrhage of various origins in which a prompt cessation of the bleeding occurs on the intravenous use of 15 to 20 cubic centimeters of 30 per cent sodium citrate. In line with this idea one would feel that citrated blood might act more efficiently than whole blood in the treatment of a persistent and concealed hemorrhage that could not be controlled by other means, although in such a case I would prefer to give unchanged blood, and then administer sodium citrate intravenously in more nearly therapeutic amounts than that used as an anti-coagulant. I merely mention the above because there is undoubtedly a justification for some difference of opinion as to the choice of methods.

No description of the citrate or indirect method is necessary, as it merely consists in allowing the desired amount of blood to flow from the donor's veins into the citrate solution, after which it is introduced into the vein of the recipient either by means of a series of syringes, or through a needle which is connected to a receptacle by a rubber tubing. Of the direct methods it may be said that there are several very satisfactory outfits on the market, or, in the absence of any of those, a series of syringes may be used. The transfusions reported in this series were done with the Scannell direct transfusion apparatus.

Before the study and establishment of the four blood groups, severe reactions, usually the result of agglutination or hemolysis, were all too common, so that the operation of blood transfusion had difficulty passing the stage where it was considered anything more than a last-resort procedure. While it is known that the four groups are not all-conclusive and it is a recognized fact that there are sub-groups, the fact remains that with careful grouping and insistence on cross-matching of the donor's and the recipient's blood, there is extremely little likelihood of serious consequences arising as a result of hemolysis or agglutination. Occasionally, in spite of grouping, fatalities have occurred. When in the case of such an accident all other possibilities have been excluded and the death has been accompanied by shock, hemolysis, and hemoglobinuria, the source of error may be any of the following:

1. Lack of skill in grouper
2. Deterioration of test sera
3. Deceptive reactions
4. Pseudo-agglutination (rouleaux-formation)
5. Deviation of group from the four classical types, with formation of new agglutinins
6. Temporary bacteriemia causing no disturbance in donor.

The American Society of Immunologists considers thirty minutes at room temperature suf-

ficient to show agglutination in a test of blood for grouping or matching. Shera, in a study of a fatal case of suppression of urine following a blood transfusion, found no agglutination at room temperature at the end of thirty minutes, but found on repetition that it occurred at the end of sixty-five minutes. He now incubates the slides at 37 degrees centigrade from 60 to 75 minutes, placing them in Petri dishes lined with moist filter paper. Opitz, in transfusions in children, makes a preliminary injection of from 20 to 30 cubic centimeters of blood and then pauses to note any evidence of reaction due to incompatibility. In spite of this precaution there was, in his series of 200 transfusions, one death from hemolysis. Two much stress cannot be put on the paramount importance of careful grouping and matching.

When one considers the causes of the reactions which more or less do follow in the wake of this procedure, one feels that in general the more skilled the operator the fewer in number the reactions will be. Lewisohn in an article on citrate transfusions states that when blood transfusion was taken out of the hands of inexperienced operators at Mount Sinai hospital the incidence of reactions was reduced from 34 to 8 per cent in the citrate, and from 23 to 13 per cent in the Unger method. He feels that many of the reactions seen in the citrate method are due to a chilling of the blood which takes place while it is being collected. Mueller tabulates the causes as follows:

I. Technical
   1. Too rapid transfusion
   2. Partial destruction of red cells in passage through needle
   3. Formation of small clots
   4. Toxic action of citrate

II. Biological (Important)
   1. Improper grouping and matching.

Lewisohn contends that reactions are never due to citrate and submits in defense the work of Newhof and Hirschfield, who injected 6 to 8 grams of sodium citrate intravenously without causing a reaction. However, as was stated above, citrate has a directly opposite action on blood in a test tube to that in the body, so this does not appear to be a valid argument. In my own series the reactions in the direct transfusions were markedly less than those occurring in the citrate method, but whether this was a result of this single factor or not is impossible to say. I believe that when all other factors are eliminated as possible causes there are still a small number of reactions left due to citrate alone, although I believe it is true that reactions from this source will never prove serious. Rolleston gives three types of reactions, as follows:

1. Agglutination and hemolysis — occurring immediately
2. Proteolytic—delayed 1 to 2 hours
3. Systemic or constitutional reactions.

Shera reports one, and Blaker and Dodds two cases of uremia due to mechanical blocking of the kidneys and liver with blood cell agglutinations. Bancroft reported a case of anuria following a transfusion for the relief of uterine bleeding in which 400 cubic centimeters of blood were given. Anuria followed the first day following the transfusion, with one ounce of urine being passed in twenty-four hours and an increase in the creatinin and blood urea. Complete decapsulation of both kidneys resulted in the recovery of the patient.

Oehlicker divides the reactions into two groups, the immediate ones occurring in the first three minutes and accompanied by shock phenomena and later by hemolysis and hemoglobinuria, and those occurring later which he says are due to an idiosyncrasy of the patient to the newly introduced serum. It is quite possible to have a reaction in a patient receiving blood from a donor who has safely given blood to the patient before. One explanation of this phenomenon can be found in the fact that sometimes the blood of a patient is so depleted that the agglutinins are not present in sufficient amounts to cause agglutination, but after the first or second transfusion these substances may have increased to the point where they give a reaction with the blood of a donor whose blood had previously been used with safety. This is merely another argument for cross-matching every time a transfusion is done, regardless of the donor. It is also a well-known fact that an individual's group may be altered by having had his blood mixed with that of one or more donors, particularly when the blood used has been of another group.

The amount of blood given depends of course upon the condition for which it is being given, and the size and weight of the patient. With the exception of those cases of acute hemorrhage with massive loss of blood, and exsanguination-

transfusion, the tendency is towards smaller amounts given oftener. It is our practice to give, unless there are indications to the contrary, in the neighborhood of 500 cubic centimeters to the average adult. Copher in a study of 245 cases reports as much as 2,000 cubic centimeters given at one time. In hemorrhage of the newborn Le R. Brooks advises amounts of from 100 to 200 cubic centimeters. Opitz gives infants from one-fifth to one-third the total blood volume. It has been our practice to give infants and small children approximately 15 cubic centimeters per kilo weight. Holbertisma states that the change in the number of corpuscles depends in the main on body weight and the quantity of blood given, and that in general an increase of 1 million red cells depends on the use of 15 cubic centimeters per kilo of body weight. Moore and Dennis, in the use of citrated blood subcutaneously, give 50 cubic centimeters per kilo in newborn infants and up to one year of age. At two years of age 25 cubic centimeters is the optimum. They use 90 cubic centimeters of blood and 10 cubic centimeters of a 2 per cent citrate solution, and make a point of introducing it subcutaneously, and not intramuscularly.

Our own series of 150 transfusions comprise both citrate and direct methods, the latter in preponderance, since once having taken up the direct method one is loath to return to the citrate. The entire series was done on eighty-three patients, fifty being females, and thirty-three males, with a number of infants in each group. The largest number of transfusions done on any one patient was five. A list of the conditions for which the transfusions were given, together with the result when the hospital stay was terminated, is given below:

	Well	Improved	Died	Total
Pernicious anemia	0	11	3	14
Secondary anemia	1	10	0	11
Duodenal or gastric ulcer	1	4	2	7
Carcinoma of intestinal tract	0	2	4	6
Menorrhagia	4	2	0	6
Septicemia	2	0	3	5
Postpartum hemorrhage or shock	3	1	0	4
Postoperative hemorrhage or shock	3	0	1	4
Acute mastoiditis	2	1	1	4
Thrombocytopenia	1	1	2	4
Pelvic inflammatory conditions	2	1	0	3
Myelogenous leukemia	0	1	1	2
Lymphatic leukemia	0	0	2	2
Uterine fibroids	1	1	0	2
Puerperal anemia	1	0	0	1
Carcinoma of bladder	0	1	0	1
Carcinoma of prostate	0	0	1	1
Subacute bacterial endocarditis	0	0	1	1
Hemophilia	0	1	0	1
Rupture of liver	0	0	1	1
Ulcerative colitis	0	1	0	1
Pyemia	0	1	0	1
Carbuncle of upper lip	0	0	1	1
	21	39	23	83

There were in the series twenty-eight transfusions using sodium citrate as an anticoagulant. Among these there were fourteen reactions, or 50 per cent. In the remaining 122 transfusions in which unchanged blood was used there were twenty reactions, or 19.7 per cent. In the series in which cross-matching was used in addition to the grouping, the reactions occurred in 11.1 per cent of cases. All rises in temperature or chills following transfusions, unless such phenomena were more or less regular occurrences, were interpreted as reactions from the transfusion. From these figures it appears that the incidence of reactions has been reduced by using fresh, unchanged blood, and still further reduced by careful cross-matching in addition to typing.

The various groups were encountered as is shown in the following table:

Group I	3 cases
Group II	35 cases
Group III	12 cases
Group IV	33 cases

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THE MEDICAL RESERVE

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IN the brief time at my disposal it will be necessary to consider only the problems of the Medical Reserve Corps without considering their relation to pacifism, etc. The problem of National Defense is your responsibility, just the same as it is that of every other citizen. Preparation for national defense holds the same relation to national security that life and fire insurance do to the family, police and fire department to the city, and state police to the state. Our present state of development does not yet uphold the Utopian ideal that life and wealth are perfectly safe anywhere.

Following the close of the World War, Congress saw fit to adopt the present policy of National Defense in the year of 1920. The need of such preparation was amply demonstrated after the mobilization of 1917.

The land defense force now consists of the Army of the United States, often called the three in one Army. It is composed of one part Regular Army, two parts Militia, and three parts Reserves. It is not the wish of the American people to have a large standing army, but rather to depend upon its trained citizens. The Regular Army is now acting almost entirely as instructor for the other components.

Three sources of candidates for the Reserve are available:

1. High school boys and others of 17 to 24 years of age, who are trained for one month at the summer Citizens' Military Training Camps. The boys are not paid, but all expenses are taken care of by the Government. Every safeguard to moral and physical health surrounds these boys. These camps also help to solve one of the modern problems of occupation for boys during vacation time.

2. The Reserve Officers Training Corps, consisting of university and college men. In some schools the course is compulsory, in other voluntary. During the college course one period of six weeks is spent in training camps. These young men receive some compensation during the college year and are paid while in camp. It is from this group that most of the officers for the Reserve will be drawn in the future.

3. The Officers Reserve Corps, consisting of officers who have had service during the World War or who have been since appointed in the various grades; either from the Reserve Officers Training Corps, or directly from civilian life. National Defense requires a certain amount of service from all able-bodied male citizens, and it is probable that in case of another national emergency not only all the men, but all the women and older children would also be required to serve in some capacity. This is an obligation imposed upon every citizen to compensate to some extent for the many privileges we enjoy as citizens of this great country.

Naturally, in such a plan the medical profession plays a large and important part. The doctor is one of the first men to go into action and about the last out. You recall the mobilization of 1917, and how the doctors were called upon first, in order to select the physically fit and eliminate the unfit. You may also recall that during this mobilization it was necessary to assemble a large number of officers for training before the drafted men could be called into camp, as we were so totally unprepared to handle an army. This training program which we are now carrying on is designed to obviate such a recurrence in case of another mobilization.

Right here I wish to call attention to one thing on which the Medical Department hopes to improve its record in the future. We failed to eliminate all the mentally unstable, and as a consequence we now have a tremendous problem that the Veterans Bureau is handling and will continue to handle for many years. It is a distinct challenge to the Medical Profession to develop a method that will prevent the recurrence of such a disaster in case we should ever be called upon to meet another national emergency. We must find some way of eliminating the mentally unfit. A person may be handicapped physically in many ways and still be useful, but if he is mentally unstable he has no place in the

National Defense, and not only no place, but is a burden to all concerned. I therefore invite your serious consideration to this problem of proper classification of the mentally incompetent. It must be properly solved, not only in case of war mobilization, but for the peace time industrial mobilization that is constantly in progress, where large numbers of competent men must be gathered together. It is now especially necessary, as the urge of still greater speed appears to be at hand, and if any man should be nervously stable, it is the man who is controlling an airplane. So, the problem is not one entirely for the military, but has a very strong bearing on civil life.

Many of our doctors object to the training offered by the Reserve Corps on the grounds that a medical man does not need it. While it is true that a certain amount of the medical military work can be done without special training, there still is a greater amount of it which requires much special work in order to be fitted to handle the medical department problems efficiently. Of course, a well trained surgeon can step into the operating room of a military hospital and carry on. The same is true as far as the medical wards are concerned, but this is about as far as the routine training of civil life will successfully operate. We have the same problem in the medical department on a somewhat smaller scale that the line troops have, *i.e.,* we must select our men, train, feed, clothe and shelter them. We must be able to transport them, guided only by a map oftentimes; and know how and where to get supplies for our work; so it is necessary that every man that serves with the army should have certain basic training. It is essential that he know something of the organization of the Army, that he may play his part. If he knows nothing of the other departments of the Army he can not properly function as part of the team.

What methods are available for acquiring this necessary basic knowledge? There are now well established correspondence courses, starting from the beginning and carrying through to the advanced medical military work. There are the summer training camps, of two weeks each summer, in which a large number of officers are assembled and who are trained in the special problems of their particular units. Regular courses of two to six weeks are given at Carlisle Barracks, especially for commanding and administrative officers.

Conference courses are also established at various places where a group of Reserve Officers can be assembled, especially where a fair number belong to the same organization, such as a General or Evacuation Hospital, or a Medical Regiment, and these groups take up the study of their special problems. And for the younger men a special Flight Surgeon's course of three months is given at San Antonio, Texas. This is a very excellent post-graduate course and fits the students for the rapidly advancing demand for civilian flight surgeons.

The Medical Department is a large organization and is comprised of the Medical Corps, the Dental Corps, the Veterinary Corps, the Medical Administrative Corps, the Sanitary Corps, the Nurse Corps and the enlisted personnel. Our active organizations are General Hospitals, Surgical Hospitals, Evacuation Hospitals, Station Hospitals, Convalescent Hospitals, Regimental Medical Detachments, and, the latest organization, the Medical Regiment. It is therefore essential that a doctor who is to take up the work in the Army should know something of his own department at least and its relation to the rest of the Army.

In order to convey to you as vividly as possible what certainly would be the problems of some of you in case of mobilization, let us assume that a great national crisis exists and that mobilization is ordered. How big is the job ahead of us?

I stated that the Army is now on a one-two-three basis: one part Regular, two parts National Guard, and three parts Reserve. Our present mobilization plans provide for six field armies, and this means nine Regular Army Divisions, eighteen National Guard Divisions, and twenty-seven Reserve Divisions. Roughly, a Division is 20,000. A simple problem in arithmetic shows:

9 Divisions Regulars	180,000
18 Divisions National Guards	320,000
27 Divisions Reserves	540,000
	1,040,000

These are the active fighting forces and do not include the vast numbers necessary in the supply, transportation, record and other auxiliary

departments, nor the corps and army auxiliary combat troops.

This would require approximately 200,000 officers, and of these some 20,000 would belong to the Medical Department and 16,000 would be Medical Corps. Recall further that this is not the end, but only the beginning, as in the last war we had about four times that number engaged at the time of the Armistice. Is it probable that a problem of this magnitude can be attempted with any chance of success without extensive preparation over a long period?

But, you may object, the doctor's business is to get people well, and will he have more to do with all this mobilization than to care for the sick and injured? Yes, vastly more. During peace times in well organized hospitals, we merely notify the proper authority that we will operate and when, and everything is ready for us. Did you ever work out all the details of just how this very technical preparation comes about? As your work is in the professional domain and not in the executive, it would not be suprising if you had not. But such perfection of administration does not just happen. Someone is on the job long before you step into the operating room.

In war time the Medical Department will have to organize and handle its own department and woe be to our country if we fail on the job. Nothing does more to maintain the morale of a command than the knowledge that if injuries do come an efficient medical service is at hand to give prompt and skilled relief. But how are we to do this if someone can not organize, equip and manage the relief agencies such as the Evacuation and Surgical Hospitals far up at the front, cannot provide and maintain an efficient and rapid evacuation such as our Medical Regiments are designed for, provide definite hospital treatment at the general hospitals, administer Convalescent Camps and finally see that the injured and broken down reach home safely? All of these are Medical Department functions. You will not find these units organized, equipped and ready to serve unless you do the work.

Suppose for example, you are the Commanding Officer of General Hospital. This organization has 42 Medical Department Officers, 120 Nurses, 400 Enlisted Men. With this force you are expected to handle a 1,000 bed hospital in all of its departments and in a crisis be able to nearly double that capacity for a time. You are in a foreign country. Buildings of the shack type are available. Supplies for the unit are in box cars on a side track four miles away. Motor trucks are turned over to you. You are directed to prepare a place for your nurses, who will arrive in two days; to be ready to receive patients from the front in four days. What would you do? If your organization is trained and each man knows his job, the task is not at all impossible; but with an untrained and undisciplined group nothing but chaos could result. It is the answer to such problems as this that we work out in camp.

To make the problem still more vivid, consider for a moment what might happen right here in St. Paul, if a national crisis similar to that of 1917 should recur. As Corps Area Surgeon of the Seventh Corps, *i.e.*, Minnesota, Iowa, Missouri, Arkansas, North and South Dakota, Nebraska, and **Kansas, it is my job** to know something of the medical resources of these particular states, but this discussion will apply only to Minnesota.

The last American Medical Association directory shows the population of Minnesota as 2,387,125; of this number there were 5,806 doctors of medicine, 3,328 of this number were members of the American Medical Association. In the state there are 233 hospitals with a bed capacity of 24,551, with an average patients hospitalized, 19,778. The hospitals in your state may be classified as follows: Individual, 62; independent, 44; church, 47; state, 19; county, 15; city and county, 4; city, 13; partnership, 17; fraternal, 5; Indian, 4; Veterans Bureau, 1; industrial 1; and Army, 1. At the time of this survey there were approximately 267 medical reserve officers in the state of Minnesota. Briefly, the above figures will show that the ratio of doctors to your population of the state is 1 to 411. The ratio of doctors, members of the American Medical Association, to the population is 1 to 719. And the ratio of doctors in the Medical Reserve to Doctors that are members of the American Medical Association is 1 to 12.5.

If this country is called upon to mobilize its man power for major effort in the event of war, your state would probably be called upon to furnish upon M day (which means day war is declared), until a period of probably 120 days thereafter, approximately 60,000 men. Many of you know, as a result of our last war, what a task

this would mean for the Medical Department. Our duties and responsibilities in the Medical Department are specifically laid down in Army Regulations. It may be tersely said that it "consists of conservation of the man power of this country." In my opinion, our task is greater on M day than any other branch of the service. Immediately we must perform physical examinations in the case of all voluntary enlistments and later of the draftees at the reception centers established throughout the country. In your state, such a reception center would probably be placed at Fort Snelling.

Medical personnel would be also required for special training battalions to care for those found suitable for "limited service only," when their defects are considered curable. Probably the present station hospital of Fort Snelling would become the reception center hospital which would naturally have to be expanded to meet the situation. The Crane General Hospital at St. Paul, an inactive Regular Army Unit, with a capacity of 1,000 beds, would immediately be placed in operation.

Station Hospitals would also be stationed elsewhere through the state provided the number of troops mobilized and distances from St. Paul and Minneapolis indicated such a procedure desirable. Our plans as a whole, roughly, means that our requirements would be 92 medical officers in St. Paul, and 2,100 beds for Corps Area overhead troops. Medical Officers for the Corps Area service overhead, in addition to the Medical Department personnel required for the units mobilized in the state of Minnesota, approximately 684; making a total of 776. We said previously, that at the present time there were 267 Medical Reserve Officers in the state of Minnesota, and now I have shown you the probable needs in the way of Medical Reserve Officers on M day, or shortly thereafter, in order to conserve the man power of our army in time of war.

While there are many active and interested Medical Reserve Officers in your state, these figures readily illustrate just how far the medical profession lacks in its part in the preparation for National Defense. These figures that I have given you are very conservative and have not included those officers sent to training schools or required as an overhead service command by the War Department. It is believed that all able-bodied doctors not in the Reserve Corps on M day would be classified as student candidates and would probably not be commissioned until graduated from training schools. This naturally means that the doctor of medicine not a Reserve Officer can not participate in our big problem at a time when he is vitally needed.

In addition to the hospital units required by Corps Area service overhead in case of national emergency, the following hospitals would probably be mobilized in the state of Minnesota: Ten General Hosptals, 1 Evacuation Hospital, 1 Surgical Hospital, 1 Convalescent Hospital, 1 Hospital Center and 1 Medical Laboratory. This does not include the medical personnel attached to line organizations.

The discipline, training, feeding, housing and morale of a command demand thought and study. These problems are likely to be forced upon any one of us in time of serious stress, and it is to handle such emergencies that the special training for Medical Department personnel has been instituted. We do not expect that you will become past masters of all the arts of command, but we do know that the fact that you have thought on the subjects, have been in camp, seen some of these problems worked out, and have a mental picture of the tasks involved, will make for much greater chance for success than could possibly be the case where no thought has been given to such problems.

I am particularly anxious to have the medical profession consider this as its own problem. Practically every doctor will be required to serve in some capacity in case we are ever unfortunate enough to be drawn into war again, and he will naturally be of much more value if he knows something of the duties of a medical officer in time of action. These men will necessarily be drawn from the ranks of the Medical Society to a large extent. Whether you are of age for active service or have passed that period, it is a matter that should claim your interest, as this is your country, and the army and national defense program is approved by you through your representatives. It, therefore, come right back upon you. Each of us must play his part. You may not be able to take an active part, but you can take an active interest and help the young men who are coming on, and who so frequently appeal to the medical men for advice.

The training in camp is not without compensation, as it brings men from all parts of the coun-

try into contact and very pleasant associations are formed. Camp is of great value to the medical man as a complete and radical change of thought and work, and hence a most valuable vacation. His application of professional knowledge to military problems starts unused gray matter to working and rests that which is so continually active during the other fifty weeks. We have in the Medical Reserve Corps today many of our most prominent medical men, practically all the specialties being represented. These men give much of their time and thought to the promotion of National Defense and are making a most valuable contribution to the safety of their country. I believe it the duty of every doctor of whatever age or line of practice, to be informed upon the subject of National Defense, especially as it pertains to the Medical Department, and to be ready to do his part, whatever it may be.

THE BATTLE OF STATISTICS

In a recent number of *Science* Professor C. H. Forsythe, of Dartmouth College, crosses swords with the optimists on the subject of the increase in the length of life. He says that the purpose of his paper is to "take issue with those who are so elated with results obtained in their own immediate fields leading to significant reductions, not only in certain death rates, but also in the prevalence of certain diseases, that they feel justified in predicting marvelous increases in the average length of life in the no great future." "Most of these optimistic authorities have failed to appreciate that practically all these results have been obtained in children's diseases, and very little or no attention has been given to the ages beyond the prime of life."

The writer claims that conditions in this country at advanced ages have been on the down-grade ever since the first mortality records were established, and we have now reached such a pass that all the great gains at the early ages are already more than offset by the losses at advanced years. In brief, he considers that he can show that the average length of life in this country is now actually decreasing. "It is most momentous and almost inconceivable that a civilized and even a highly prosperous nation should have a decreasing average length of life." This sad news has been echoed from some of the insurance companies, showing that in the older age groups no prolongation of life was observable. The great decline at advanced ages is concentrated about age 70, although it extends as far back as the age 40, according to Professor Forsythe.

"The whole picture points consistently and inevitably to a future of a declining length of life until the American adult wakes up to the fact that the odds are at present heavily against his living as long as his father or grandfather." Some will say that this is all the natural consequence of the great migration to the cities; others, that life has become too fast and strenuous and that we do not yet know how to adjust ourselves to such a life. To physicians the great problem will loom as one of relieving strain upon the heart or conquering cancer, but little will be accomplished until the American adult himself is duly informed and made to realize that he is in the midst of a losing fight and that the situation will continue until he applies himself energetically to be superior to his environment. Medical authorities can be depended upon to care for the children and their diseases, but they have little or no chance to interfere with the lives of adults. There is surely no worse influence than that wielded by the well meaning individuals who go around airing their ill-founded beliefs that all is going well and before long everybody is going to live 75 or 100 years.

All this in July 26, 1929, but in the September 13th number of the same magazine, Mr. J. D. Ryan, of San Francisco, in rebuttal, states that "mortality statistics are not a satisfactory basis for such generalizations." "The great reduction in infant mortality and in deaths from communicable disease in recent years has postponed death to the later years, and the lives thus prolonged are usually sustained at diminished vitality." It is reasonable to expect that the average general health of the community has been diminished, and the average death rate during subsequent years is increased by these extended lives. If a substantial number of these previously short lives has extended to 40 and 50, the average age of death of all persons passing the age of 40 is also reduced, and the ratio of the number of persons reaching the age of 50 to the total population will also be reduced. The increasing mortality due to certain organic diseases is frequently referred to in current discussions as indicating a greater susceptibility induced by changes in our manner of living or by the devitalizing influence of organic stresses incident to more strenuous activities. "There is absolutely no foundation in fact or experience for such beliefs." More people escape death by infections than formerly, but as all of us ultimately die, it is inevitable that there should be a corresponding increase in the number of deaths from other causes. We have reason to expect a continued reduction in mortality from tuberculosis, typhoid, and other prevalent diseases in the future which should appreciably prolong the average span of life. "As many of these lives will be terminated later by cancer and diseases of the heart, it would be absurd to draw alarming inferences from subsequent statistical reports showing increased deaths from these causes." —Campaign Notes, Am. Soc. Control of Cancer., Sept., 1929.

MULTIPLE SCLEROSIS: A CLINICAL REVIEW AND FOLLOW-UP STUDY*

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ACCORDING to available knowledge, multiple or disseminated sclerosis is, fortunately, a rare disease, afflicting about eight to ten persons in each 100,000 of population. Except in breadth of clinical knowledge, little more is known of this disease than in the days of Cruveilhier (1835) and Carswell (1837). The pathologic end-products of the disease seem clear; the pathologic changes earlier in the disease may not be so well understood. The theories of etiology are numerous, but deal for the most part with primary degeneration, and infection or inflammation.

In a survey of some of the clinical facts of the disease, including the course and outcome, information was obtained about patients studied at The Mayo Clinic between January 1, 1915, and January 1, 1928, on whom a clinical diagnosis of multiple sclerosis had been made by a consulting neurologist. Most of the information was obtained in response to a series of questions sent to patients. A few patients returned for reëxamination. The home physicians of a few were kind enough to send detailed reports. From these combined sources, a follow-up study was obtained from 529 patients, which is slightly more than half of those on whom a diagnosis of multiple sclerosis was made in the period designated. An attempt is not made to give the incidence, variety, or sequence of symptoms and signs in this group during their course. It should suffice to say that a disease which produces multiple lesions or plaques of sclerosis or fibrosis in various parts of the central nervous system, more especially in the white matter, can produce a great many and varying combinations of symptoms, among which one may see the triad of Charcot: scanning speech, intention tremor, and nystagmus. As a matter of interest, certain comparisons will be made between this group and a large group of patients who have had epidemic encephalitis.†

These patients came, for the most part, from Minnesota and adjoining states and provinces. Evidence for hereditary transmission of the disease is meager. However, in seven cases there was apparently a similar disease in near relatives. In five cases a brother or sister had a like disease. In one case the father of the patient probably had multiple sclerosis. Several members of one family had a syndrome that was much like multiple sclerosis. The sexes seem about equally affected; there were 277 males and 252 females in this group, making the ratio of males to females 1.1:1 (the ratio of males to females is 2.5:1 in epidemic encephalitis, and 2.8:1 in paralysis agitans so diagnosed prior to 1917.) The disease had an insidious onset in 456 cases (86.2 per cent), which is in strong contrast to epidemic encephalitis, in which the history of an acute onset was elicited in 84.8 per cent of a large group of patients studied. The disease makes its appearance most frequently in the second and third decades of life. There are well authenticated cases in which the onset was in infancy or early childhood, and also in well advanced years. The data of the age of onset for this group are presented in Figure 1.

The disease may make its appearance at any season of the year, but I have felt that the onset, and recurrences, are more frequent in spring and early summer. The seasonal variations in the following case are of interest.

Case 1.—A man, aged twenty-three years, came to The Mayo Clinic in November, 1927, complaining of difficulty in walking, which first had made its appearance in April, 1918, and which had progressed to almost complete paralysis of the lower extremities in two weeks. His hands also had been weak and numb. There had been some loss of sphincteric control. Within six weeks from the onset, he had improved markedly and had been up and about, but had known his arms and legs were not right. In January, 1920, within two weeks of the onset of the trouble, he again had become even more paralyzed than he had been in April, 1918. There had been complete loss of sphincteric control. By June or July, 1920, he had been walking, but his legs had been stiff. Each year since, usually in April and November, he had had marked exacerbations in his inability to walk or to control his sphincters. These

*Read before the Minnesota Neurological Society, Rochester, Minnesota, June 8, 1929.

†Ziegler, L. H.: Follow-up studies of persons who have had epidemic encephalitis. Jour. Am. Med. Assn., 1928, xci, 138-141.

spells had lasted for a few weeks. His condition had become much worse two weeks before he came to the clinic. He had had difficulty in controlling his bladder and rectal sphincters.

Examination at the time of the patient's appearance at the clinic revealed the following: both vertical and horizontal nystagmus, bilateral signs of lesions of the pyramidal tract in arms and legs, ataxic and spastic gait, slight incoördination of arms and hands, absence of

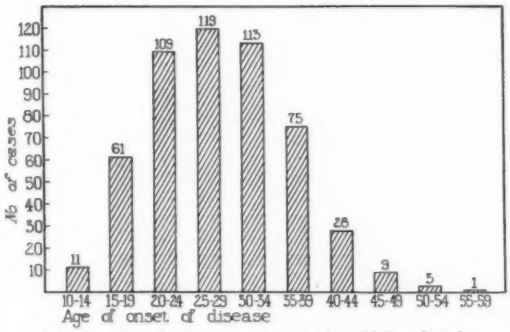

Fig. 1. Age of onset in 529 cases of multiple sclerosis.

left lower abdominal reflexes and some loss of joint and vibration sense below and including the crests of the ilia. Bladder and rectal sphincters were poorly controlled. Examination of the spinal fluid disclosed five cells in each cubic millimeter, a negative Wassermann reaction, a negative test for globulin, and prompt response to compression of the jugular veins, with slow return to normal.

That the symptoms of the disease tend to undergo remission and recurrence is common knowledge. Complete remission of all symptoms would seem to be rare, but it does occur. On the other hand, complete remission of any one symptom or sign is not unusual. Remissions may be striking. A patient may become nearly helpless, and within a short time his condition may return almost to normal and he may be able to work. A patient is likely to praise any remedy which he happens to take during a remission and to condemn one taken while a recurrence is in progress. As a rule, remissions become less and less complete and recurrences more and more pronounced. These facts make the evaluation of treatment difficult. That long remissions do occur is illustrated by the following case.

Case 2.—A married woman, aged forty-nine years, came to The Mayo Clinic in 1925 on account of paralysis of the lower extremities of four years' duration. She had married at the age of twenty-four years and had had three children, who were entirely well. At nineteen years of age the entire right side of the body had been numb for four or five months. She had not been able to write with the right hand; it had been tremorous. She had been unable to speak clearly. She had recovered so far as subjective symptoms had been concerned and had been well until the age of thirty-four years, when, during the month of May, she had begun to drag the left leg, and both legs had become numb. She again had recovered and had been well until the age of forty-five years, at which time the legs and feet had become heavy and weak and the abdomen had felt as if a band were tied about it. The lower extremities have become increasingly more spastic and useless. She has had incontinence of the bladder at times since the age of forty-eight years.

Examination at the clinic revealed all types of nystagmus, marked weakness, and slowness and spasticity of the lower extremities, making walking impossible. She had marked loss of joint and vibration sensibility in the lower extremities and slight loss in the arms. There was absence of the abdominal reflexes, and signs of lesions of the pyramidal tract could be elicited in the right hand and in both feet. The Wassermann reaction of the spinal fluid was negative and the fluid contained many erythrocytes, probably introduced at the time of puncture.

A common precursor of the disease is fatigue and a state of reduced capacity to work, which may exist for a few weeks to several years before more specific symptoms or signs make their appearance. Fatigue and reduced capacity for work also are present in many cases during the so-called remissions. These symptoms often lead to a diagnosis of neurasthenia. It is often possible, although not always easy, to collect enough evidence by a thorough neuropsychiatric examination to distinguish between the fatigue as an expression of psychobiologic inadequacy and that due to disease of the nervous system. The earliest symptoms or signs noticed by these patients are recorded in Table 1.

In more than 20 per cent of the cases the first symptom noticed was weakness, awkwardness, or inability to use the legs properly in walking. This symptom led many patients to consult orthopedists to see if "broken arches" or other disease of the bones and joints might not be the cause of the symptom. Those who complained of numbness, tingling or paresthesia of the lower extremities not infrequently assumed their trouble to be "rheumatism" and were often treated for this condition. Although pain is not a common symptom at the onset, it is not uncommon in the subsequent stages of the disease, when increased muscular tone and contractures are doubtless the cause of distress. Although a few patients consulted neurologists early because of fear of paralysis, general practitioners of medicine and orthopedists doubtless see many

TABLE 1

EARLIEST SYMPTOMS OR SYMPTOMS AT ONSET NOTED BY 529 PATIENTS WITH MULTIPLE SCLEROSIS

	Cases
Motor disturbance, both lower extremities*	116
Motor disturbance, right lower extremity	52
Sensory disturbance, both lower extremities**	50
Defective vision (blurring)	44
Motor disturbance, left lower extremity	34
Diplopia	32
Sensory disturbance, right upper extremity	20
Sensory disturbance, both upper extremities	17
Motor disturbance, right upper extremity	15
Sensory disturbance, all extremities	15
Sensory disturbance, left upper extremity	15
Loss of control of sphincter of bladder	14
Sensory disturbance, right lower extremity	13
Motor disturbance, both upper extremities	9
Motor disturbance, right upper and lower extremities	9
Sensory disturbance, left lower extremity	9
Motor disturbance of all extremities	8
Vertigo	7
Sensory disturbance, left upper and lower extremities	7
Difficulty in talking and swallowing	5
Motor disturbance, left upper extremity	4
Motor disturbance, left upper and lower extremities	4
Sensory disturbance, right upper and lower extremities	4
Rectal incontinence	4
Sexual impotence	3
Sudden loss of hearing in one ear	2
Sensory and motor disturbance, right lower extremity	2
Sensory and motor disturbance, both lower extremities	2
Motor disturbance, left upper and right lower extremities	1
Sensory disturbance, right upper and left lower extremities	1
Motor disturbance, right lower extremity; sensory disturbance, both lower extremities	1
Motor and sensory disturbance, left lower extremity	1
Motor and sensory disturbance, right upper extremity	1
Motor and sensory disturbance, all extremities	1
Sensory disturbance, right side of face	1
Weakness, right side of face	1
Weakness, left side of face	1
Girdle sensation	1
Motor disturbance, right upper extremity; sensory disturbance, right lower extremities	1
Tinnitus	1
Frequent fainting	1
Total	529

*Loss of control, awkwardness and weakness.

**Tingling, numbness, prickling, pain, burning, "electric sensations," and so forth.

patients with multiple sclerosis before neurologists see them. It behooves orthopedists, especially, to be familiar with this syndrome to prevent misdirected treatment and faulty prognosis.

Defects in vision, such as diplopia and central scotoma (resembling retrobulbar neuritis), constituted the symptoms at onset in 14.4 per cent of patients in this group. In the course of the disease, a much larger proportion of patients have symptoms referable to the eye. This fact makes it essential for ophthalmologists to have a thorough understanding of the disease. Likewise, the urologist can solve some worrisome problems for himself if he keeps in mind that the symptom of onset in a few cases of multiple sclerosis is loss of control of the bladder sphincter.

Two patients experienced sudden loss of hearing in one ear. Seven had fairly severe vertigo. The eighth cranial nerve is involved much less frequently than the second. Weakness of facial muscles was observed twice in this series, necessitating differential diagnosis between Bell's palsy and multiple sclerosis. In a few cases, objective sensory changes were such as to make the exclusion of tumor of the spinal cord difficult. In such cases, careful studies of spinal fluid and especially of pressure, by accurate methods of measurement, are of greatest importance. Although hemiparesis or hemiplegia may occur with multiple sclerosis, it is apparent from the data in Table 1 that these are so infrequent as to make it imperative to rule out other diseases before attributing them to multiple sclerosis.

Circumstances or diseases which patients associated with the onset of their condition are given in Table 2.

The onset is associated with pregnancy and parturition in many cases. Although these conditions cannot be regarded as causes in the present state of our knowledge, they favor the progress or development of multiple sclerosis. The same might be said of influenza. In the matter of injury it is difficult to separate cause and effect, especially in view of the medicolegal possibilities that develop subsequent to the appearance of a disease intimately associated with an accident or injury. The most that one could say is that a vast number of victims of accidents never have multiple sclerosis. The association of lethargic encephalitis with multiple sclerosis, in two cases, may be accidental and should not be regarded as of etiologic significance without

TABLE 2

DISEASES OR CIRCUMSTANCES WITH WHICH PATIENTS ASSOCIATED THE ONSET OF MULTIPLE SCLEROSIS

	Cases
Pregnancy, childbirth, miscarriage	25
Influenza	22
Injury to various parts of the body (electric shock in 1 case)	22
Acute illness, unnamed (fever, headache, malaise)	9
Common colds	4
Diarrhea	4
Smallpox	4
Mumps	2
Tonsilitis	2
Epidemic encephalitis (lethargic form)	2
Osteomyelitis	1
Diphtheria	1
Neuritis	1
Infected teeth	1
Alcohol (excessive use)	1
Septicemia	1
"Milk leg"	1
Sore eyes	1
Thyroidectomy	1
Herpes	1
Acute rheumatic fever	1
Arsenic or lead poisoning	1
Swelling of feet	1
Miner's nystagmus	1

much more opportunity to study the pathologic changes in both diseases.

Actual syndromes of disease found in association with multiple sclerosis are presented in Table 3. That patients with multiple sclerosis are often euphoric and happy, out of all proportion to the nature of the profound disabilities which may be present, is a frequent observation and probably accounts for the confusion of this disease with hysteria in some cases. It is not surprising, therefore, that psychosis, especially of the manic-depressive type, occurs among them. On careful clinical analysis, this fact may prove to be more than mere accident or coincidence. The other diseases found associated may be coincidental.

Of 529 cases in which data were obtained, death has occurred in 136 (25.7 per cent). The average period from the onset of the disease until death is slightly more than seven years in this group (in 112 cases in which data could be obtained). Although data from necropsy are available in only a few of these cases, the clinical causes of death, as far as could be determined, are recorded in Table 4.

TABLE 3

SYNDROME ASSOCIATED WITH MULTIPLE SCLEROSIS*

	Cases
Psychosis (chiefly manic-depressive)	6
Grand mal	2
Diabetes mellitus	2
Trifacial neuralgia	2
Paroxysmal tachycardia	1
Hemorrhagic purpura	1
Aplastic anemia	1
Psoriasis	1
Duodenal ulcer	1
Endocarditis	1
Asthma	1
Thalamic pain syndrome	1
Hysteria	1

*Foci of infection, such as are found in teeth, tonsils and the prostate gland, in these patients, would seem to be about the same as in any other group of patients of the same age without multiple sclerosis.

TABLE 4

CLINICAL CAUSES OF DEATH

	Cases
Unstated	60
Multiple sclerosis	37
Pyelonephritis	7
Pneumonia	5
Chronic septicemia	5
Influenza	3
"Stroke"	3
Malignant growth	3
Operation (during or soon after)	3
Heart disease	2
Acute intestinal obstruction	2
Tuberculosis	1
Strangulation	1
Convulsions	1
Hyperpyrexia	1
Accident	1
Suicide	1
Total	136

In a few cases, the cause of death could not be definitely determined at necropsy. It would seem probable that pyelocystonephritis might be the cause of more deaths. Septicemia from decubital ulcers occurs. Pneumonia from aspiration of food into the bronchi occurs in a few cases. The profound debility which develops in some of the cases favors intercurrent infection.

The results of examinations of spinal fluid vary somewhat, depending on technic. Probably the cell count, regardless of type of cell, would be subject to less deviation than any other test. In Table 5 are given the number of cells in each cubic millimeter of spinal fluid and the number of years from the onset of the disease until the

TABLE 5

INCIDENCE OF VARIOUS CELL COUNTS OF SPINAL FLUID IN 345 CASES OF MULTIPLE SCLEROSIS

Years from onset	Cells in each cubic millimeter of spinal fluid (one spinal puncture for each patient)																																
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	24	25	26	27	28	29	30	34	35	44	46	103
1	7	6	6	7	7	3	5	1	1	4	2	3		2	1			1		2		2	1	1			1			1		1	
2	11	4	4	2	3	6	1	1	1		1	2		3	2	2																	1
3	7	7	7	2	3	2	3	3	1	1			1				1	1		1	1												
4	6	3	5	1	7	1	3		2	1			2		1		1	1	1					1	1	1							
5	9	2		1	1	2	2	1					1	1		1			1	1								1	1				
6	3	1	2	2	2		3	2			1		3	1			1																
7	6	4		2	1	4			1																								
8	4	1	2	3	1		2	1		1				2	1				1				1										
9	2	2	1	1	1	3													1														
10	4			1		1	2				1		1		1	1			1														
11	2	1	1						1																								
12	2				3			2				2				1																	
13		1	1		1																												
14	2	3		1		1											1																
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21	1																																
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23					1																												
24	2																																
26	1																		1														
Total	74	38	29	26	31	24	23	11	7	7	5	7	8	9	6	6	4	3	6	4	1	2	2	2	1	1	1	1	2	1	1	1	1

lumbar puncture was done. Puncture was done once only in each case. It will be observed that in a few cases the cell count was high many years after the onset of the disease, and also that it was low or normal soon after the onset of the disease. Data concerning cell counts taken frequently during the long course of cases of multiple sclerosis are needed.

The data relative to clinical course and occupational disability are given in Figures 2 and 3.

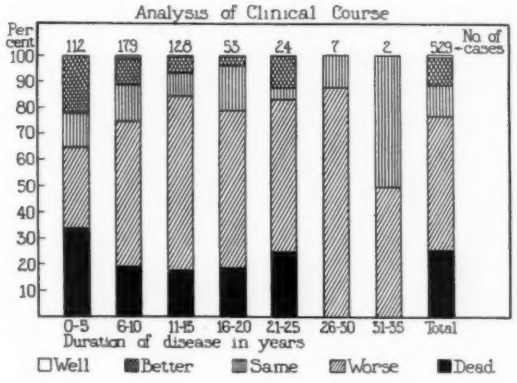

Fig. 2. Clinical course in 505 cases of multiple sclerosis.

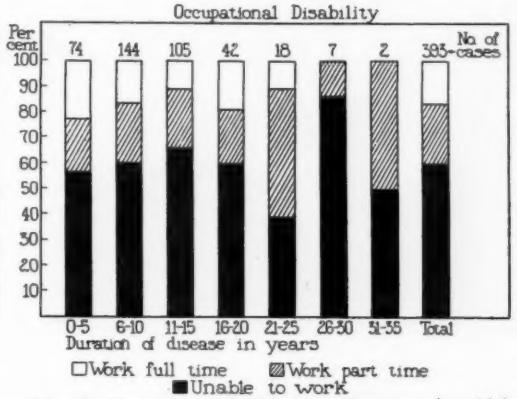
Occupational Disability
Per cent
No. of cases
74 144 105 42 18 7 2 393
0-5 6-10 11-15 16-20 21-25 26-30 51-55 Total
Duration of disease in years
Work full time
Work part time
Unable to work

Fig. 3. Occupational disability in 393 cases of multiple sclerosis.

Multiple sclerosis is for the most part a progressive disease. There are probably not less than 10,000 victims of the disease in the United States alone. Most of them, in the prime of life, are by their disease denied the privilege of economic usefulness. They not only are unable to work much, but spend what they have saved, or use the funds, time, and energy of their friends and relatives in seeking possible relief. Such are the purely economic aspects of the disease, to say nothing of the annoyances and personal discomforts of this small army of afflicted persons.

Treatment is based on trial and error. The etiology of the disease needs first to be discovered, after which treatment may be put on a rational basis. Typhoid vaccine, sodium cacodylate, salvarsan, compounds of silver, hydrotherapy, heliotherapy, rest, and good general care, all have their devotees and may be justified to keep hope alive. There is no remedy at present that one can turn to with assurance that it will arrest or retard the disease. The chief encouragements to be offered to patients are that progression may be slow, that the disease, in a few, may become arrested, and that long remissions are possible. The combined efforts of economic and scientific forces should be enlisted to the end of eradicating this among other diseases.

SUMMARY

Five hundred twenty-nine patients with multiple sclerosis were traced—and their clinical course reviewed.

Evidence for hereditary transmission is meager. The sexes seem to be about equally affected. The disease is usually insidious in onset and makes its appearance chiefly in the second and third decades of life. A season of onset is not definitely established.

Remissions of single symptoms and signs are common. Remissions of all symptoms and signs are rare. Striking and long remissions occur.

The most frequent symptoms of onset are motor or sensory disturbances of the lower extremities, and visual disturbances, including diplopia and central scotoma.

The patients associated the disease mostly with pregnancy and parturition, but a number also associated it with injury.

Patients with multiple sclerosis often have emotional disorders and are euphoric and happy in contrast to what one would expect from the severity of the disability.

Clinical causes of death, so far as they could be determined, were pyelonephritis, septicemia, pneumonia and intercurrent infection.

The disease is progressive and very disabling. The etiology, thus far, is not established. Treatment is based on trial and error and the results of some methods of treatment are hard to evaluate because of the nature of the disease. Occasionally the disease appears to become arrested, and long remissions occur.

CASE REPORTS

PRIMARY BILATERAL ADENOCARCINOMA OF THE BREAST*

REPORT OF CASE

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According to Deaver and McFarland "bilateralism in cancer of the breast is uncommonly met with, but in a majority of instances the process in one breast is secondary to a primary nodule in its fellow of the opposite side. Simultaneous involvement of both organs is extremely rare." These observers have noted eleven cases of bilateral carcinoma of the breast in 200 cases. Poulsen noted eleven cases in 355, and Schwarzkopf noted five cases in 323. Deaver and McFarland believe that bilateral tumors of the breast are more commonly benign than malignant although they do not agree with Primrose that bilateral tumors are usually benign. The case we are reporting here is one of simultaneous bilateral adenocarcinoma of the breast.

REPORT OF CASE

A woman, aged sixty-eight years, mother of seven living children, came to The Mayo Clinic first in 1924, because of attacks of vomiting. A diagnosis was then made of atypical anorexia nervosum and the patient was advised to return home. She came to the clinic a second time five years later complaining of insomnia and breathlessness of about four years' duration. She had had thrombophlebitis of one leg two years before her second visit and many years before she had had a minor plastic operation on the cervix.

The patient was florid, moderately obese, 5 feet 3 inches in height, and weighed 171 pounds. The systolic blood pressure was 180 and the diastolic 120 mm. of mercury. The pulse rate was 80. The heart was slightly enlarged (graded 1). Exertion caused dyspnea and mild cyanosis. Unknown to the patient a single tumor was present in each breast; they were associated with slight dimpling of the skin. The tumor of the right breast was in the upper outer quadrant and was approximately 3 cm. in diameter. In the left breast the tumor was just above the nipple, and was about 2 cm. in diameter. The results of roentgenologic examination of the chest were negative. The specific gravity of the urine was 1.015; it did not contain albumin, but there were 10 pus cells to a low power microscopic field.

A diagnosis was made of myocardial degeneration with loss of compensation and ascites, probably on the basis of the former hypertension; and bilateral tumors of the breast. It was decided to send the patient to a hospital for treatment of the cardiac disorder; if relief was obtained the tumors of the breast were to be excised and, if they proved to be malignant, radical amputation of the breast would be performed immediately. In the hospital the patient was given 8 grains of ammonium nitrate, three times a day with salyrgan (1 c.c. intravenously every third day) to combat ascites. The fluid intake was limited. After three weeks of treatment the patient was so much improved that she was dismissed from the hospital and allowed to go home for two weeks before the operation was performed.

With the patient under ether anesthesia the nodule in the right breast was excised and examined immediately, by means of fresh frozen sections. Adenocarcinoma, graded 3,[1] was found and radical amputation of the breast was performed. Convalescence was uneventful. Ten days later the patient was again anesthetized and the nodule from the left brest removed. The tissue was examined immediately and diagnosis made of adenocarcinoma graded 4.[1] Radical amputation was performed at once. Convalescence was again uneventful.

Pathologic Data.—Although the two carcinomas were similar in some respects they were obviously two different kinds of growths. The carcinoma from the right breast was composed of many acini filled by round or oval cells, which Broders has recently come to believe is a sign of a high degree of malignancy, in contradistinction to acini composed of cells with a tendency to columnar form.[2, 3] The nuclei of these cells were not large or conspicious and mitotic figures were infrequent (Fig. 1).

From a microscopic standpoint three chief features were noted concerning the carcinoma of the left breast: (1) solid areas of round or oval carcinoma cells with ill-defined acinar arrangement; (2) well formed acini lined by a single or double layer of cuboidal to oval-shaped cells, and (3) areas in which a single layer of carcinoma cells grew between fibrous connective tissue and was apparently compressed by it (Fig. 2). None of the lymph nodes removed gave evidence of metastasis. The single areas mentioned were the only foci of carcinoma in either breast.

COMMENT

In this case, as in every one of suspected adenocarcinoma of both breasts, there are two questions to be decided: (1) Is this a true case of two separate neoplasms, one in each breast? (2) Is one lesion metastatic?[3, 4] There are several points in favor of an affirmative answer to the first question. The tumors occurred at approximately the same time, and were of about the same size. They were not in the usual situation for the occurrence of gross metastasis from one

*Submitted for publication June 25, 1929.

Fig. 1. Adenocarcinoma, graded 3; the chief characteristic is the acini filled with round or oval cells containing small nuclei (x75).

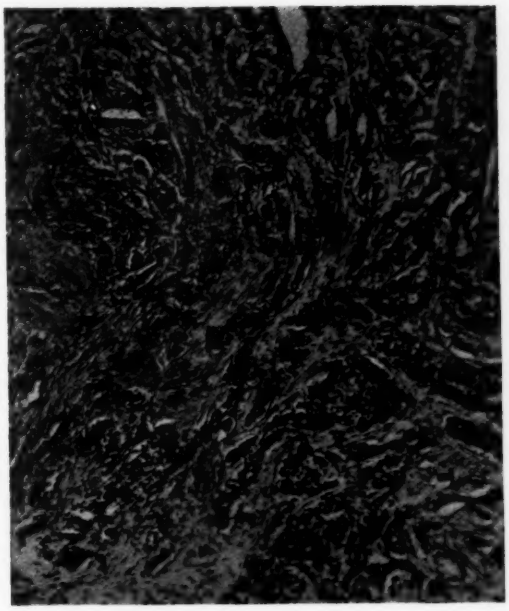

Fig. 2. Adenocarcinoma, graded 4. In the upper third of the section acini are composed of a single or double layer of round or oval cells. In the middle third of the section some of the carcinoma cells are arranged single file with much fibrous connective tissue between, whereas in the left upper corner and in the lower third of the picture malignant cells are arranged in more solid areas with less connective tissue between (x75).

breast to another, since the most common site of metastatic new growths is in the inner half of the breast, whereas these lesions were in the upper outer quadrant, and near the center of the breast a little above the nipple. The difference of the grade of malignancy is another point in favor of the bilateral origin of the tumors, for metastatic neoplasms tend to maintain the same degree of malignancy as the original lesion.[2, 5] Finally it is worthy of emphasis that the axillary lymph nodes were not involved by either tumor; this is additional evidence that the lesions were independent, since neither had apparently shown a propensity to metastasize.

The strongest argument against the bilateral origin of the tumors is the fact that metastatic carcinoma is the most common bilateral malignant neoplasm in the breast; however, these bilateral lesions most frequently develop in the second breast after amputation of the first.[3, 4]

The case reported here again illustrates the significance in surgical diagnosis and treatment of carefully investigating all tumors of the breast and of adequate treatment immediately after a definite diagnosis has been established.

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LARGE INFECTED SOLITARY CYST OF KIDNEY*

REPORT OF CASE

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Suppuration in a solitary cyst of the kidney is so very rare that every case is of interest and merits being put on record. In a hurried and most probably incomplete search of the literature I have been able to find one other case only, although more and more cases of solitary serous cysts are being reported, the number now on record approaching 200 in all.

This other single case was reported by Patel and Mallet Guy in the French Journal of Urology of April, 1925. They failed to find any other cases prior to theirs and make statement that "the classical authors do not say anything about suppuration of large serous cysts, and recent reports by Brin (1911) and Kretschmer (1920) do not give any cases." Their patient was a woman, twenty years old, married and mother of one child. She had had no urinary symptoms during her pregnancy, and had been perfectly well up to one week before. She then developed sudden pain in the left hypochondrium, with a temperature of 104.4° F. (40.2° C.). There was a painful spot with rigidity and tenderness in the left costolumbar angle, but no mention is made of any tumor at this first examination. The next day, June 3rd, a distinct swelling could be felt in the lumbar region, and this apparently increased very rapidly, for, in the next twenty-four hours, it was described as large, descending below the umbilicus and reaching in front to the mammillary line. It showed renal ballottement and seemer to fluctuate. Some pus was present in the urine. A diagnosis of suppuration, probably renal in origin, was made. Incision revealed a retroperitoneal pocket in front of the kidney, from which about a liter of pus was removed. The renal artery was injured during the operation, necessitating nephrectomy. The patient rapidly recovered and left the hospital in a month. The specimen showed the kidney to be normal except for the very large cyst which sprang from the anterior surface and which was in no way connected with the calyces or renal pelvis. A diagnosis of cyst was not made until after examination of the specimen removed. The rest of the kidney parenchyma was intact and there were no other cysts. Section of the cyst wall indicated acute inflammation, but no bacteria were found in the pus.

CASE REPORT

Miss A. N., twenty-two years old, single, a University student, a patient of Dr. Larry O. Doyle, was admitted to St. Mary's hospital Sept. 16, 1927.

Family History.—No bearing on case.

Previous History.—She had always been healthy, but, as a child in school, was proud of the fact that her "stomach" was more prominent than other children's. One year ago she complained of pain on urination with burning more intense afterwards and a stinging pain in right lumbar region. There was pus in her urine and a diagnosis was made of pyelitis due to "cold." The discomfort passed away and she was well until ten days before admission (Sept. 16, 1927), when the urinary symptoms returned. These, however, were mild and did not prevent her from climbing the mountains in Glacier Park.

Present Illness.—On September 14 she left Montana in apparently good health. About noon on September 16, while still on the train, she became nauseated and began to suffer pain in the right upper abdominal quadrant, extending down to the umbilicus and around to the lumbar region, but not to the bladder. She was nauseated but did not vomit. At St. Cloud she left the train and came to Minneapolis by auto, arriving about 11 P. M. On admission she was in considerable shock, with a pulse rate of 160, and a temperature of 99 degrees F. A catheterized specimen of urine contained a small amount of pus, and this, with the previous history, made the tentative diagnosis of acute pyelitis plausible and probable. The next morning she was somewhat better and a more careful and complete examination was made. The pulse had dropped to 120-130, temperature 101 degrees. A leukocyte count showed 32,000. The abdomen was moderately distended throughout, but in the right upper quadrant just external to the usual position of the gallbladder there was a tense, bulging, very tender swelling, about 6 or 8 cm. in diameter, extending to, but not below, the umbilicus level, and to the right, reaching the upper lumbar

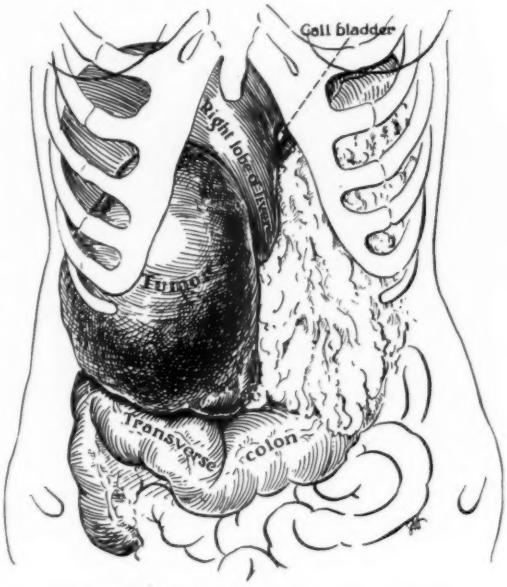

Fig. 1. Sketch made of abdomen when opened.

*Presented before the Minneapolis Surgical Society, December 6, 1928.

region, where it was indefinitely felt. Only over this mass were the abdominal muscles rigid. Upwards it seemed to be merged into the liver but auscultatory percussion showed it to be distinct from that organ. A fluoroscopy of chest and abdomen gave no diagnostic assistance, but unfortunately no films were made. She was seen in consultation by Dr. Barron on the 17th and by me on the evening of the 18th.

Diagnosis.—The diagnosis was undecided except as to the presence of pus. Four possible conditions were considered, none of which, however, quite fitted in with the clinical and physical signs.

1. Empyema of the gallbladder. Against this was the fact that the swelling did not travel toward the umbilical region as such large gallbladders usually do. Also it seemed to be too indefinite and not well circumscribed.

2. Abscess of the liver. Against this was the fact that auscultatory percussion showed it to be distinct from the general liver dullness.

3. Appendiceal abscess from rupture of a high lying appendix. I had operated on one such case in a young child, but here there seemed to be too little rigidity and tenderness for such severe clinical symptoms as were present.

4. Retroperitoneal abscess connected with the kidney. Against this was the position of the swelling, projecting the anterior abdominal wall forward, lying toward the midline, and not projecting posteriorly.

We all considered the condition as something quite unusual, and this is not to be wondered at, as a preoperative diagnosis in these large solitary cysts of the kidney has seldom been made, even when not complicated by infection.

Operation.—It was decided to open over the most prominent area, and then be guided by what exploration revealed. Dr. Doyle operated with my assistance.

On opening the peritoneal cavity a strange picture presented. A large smooth very tense tumor mass, lying close under the anterior abdominal wall but covered by the posterior-parietal peritoneum occupied all the field. The liver was pushed far up under the costal arch and was not visible, but its thin anterior edge could be felt. By lifting the lower end of the incision the transverse colon was seen, lying below the umbilical line. Toward the midline a long tongue of liver tissue reached nearly to the umbilicus. The gallbladder, stomach and duodenum were not in the field and could not be located. The tongue of liver was thought to be the left lobe but the gallbladder, which should have been to its right, was not there.

Fig. 2.

The posterior parietal peritoneum was split vertically over the tumor and was easily peeled off in front, but not from the deeper parts. The tumor could not be delivered until about three pints of pus were aspirated and its tension reduced. In getting it out of its bed the thin capsule was torn and a considerable quantity of pus was discharged on the packing. At the lower inner pole of the tumor was a hard mass which later proved to be the kidney, lying rather in front of the spinal column.

The pedicle was very large, the renal vein being the size of an index finger. After the tumor was removed the tongue of liver turned out to be a thinned-out piece of the right lobe, the normal gallbladder was to its left, and the pyloric end of the stomach and the duodenum far over in the left quadrant. The spleen and left kidney were normal.

The wound was closed up to a large drain. There was very little bleeding.

The patient reacted well, was out of bed in two weeks and left the hospital at the end of a month. A late report is that she is in perfect health and back in the University.

Pathology.—The removed tumor, minus the three pints of fluid aspirated during the operation, was

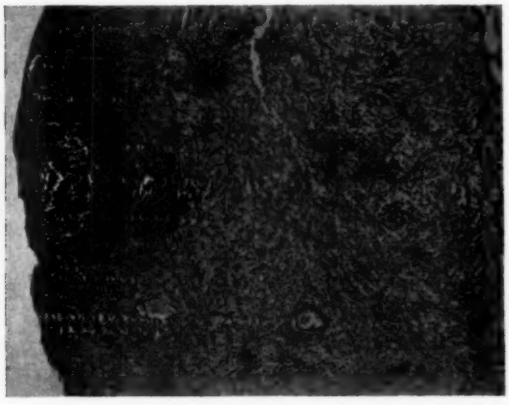

Fig. 3.

still very tense, weighed 2 pounds and was 8 inches in diameter. Estimating pints as pounds, therefore, the total weight was 5 pounds or more, and the size considerably in excess of 8 inches in diameter. The tumor was bisected, and the illustration (Fig. 2) shows half of the inner wall. The cyst springs from the upper pole of the kidney, has no connection with the pelvis or calyces, and the kidney tissue is flattened out and runs up on the wall to a point. The wall is very thin in places, and in others quite thick, varying between 2 and 5 mm. Microscopic sections through the wall show an absence of epithelial lining; leukocytic infiltration shows acute infection (Fig. 3). Section through the kidney shows, grossly, normal tissue, with no evidence of any other cyst formations. Microscopically there is a nephritis.

COMMENTS

1. The cyst was present for many years and almost certainly was congenital in origin.

2. It was not a dermoid or echinococcus, but a large solitary serous cyst which had suffered acute infection shortly before coming under our observation.

3. A point of great interest would be to discover the origin of the infection. Evidently it was not from the intestine as there was no colon bacillus odor. The history of pus in the urine and the bladder irritablity preceding the acute inflammation, and the finding of acute nephritis on section points to the kidney as the possible source.

DISCUSSION

Dr. L. O. Doyle: I haven't anything to add except that this primary infection was from the throat. She had several attacks of tonsillitis and still continued to have pus in her urine after this operation, until last spring. At that time I took out her tonsils and since then she has had no pus. She had had a cold since the time she was in the mountains.

Dr. Theodore Sweetser: I would like to mention the matter of cystoscopy on this case. I think the reason for not doing a cystoscopy on this case was the condition of the patient. There was severe shock, high fever, leukocyte count of 32,000. The patient was not in a good condition. I was not the consultant in this case, but it seems to me that, had pyelography been justified by the patient's general condition, it would have been easy enough to make a diagnosis from the pyelogram. Here is a large tumor, pushing the kidney aside, the lower calyces are rather normal in shape, and in the upper end the calyx is drawn way out and the upper part is obliterated entirely. You would have had displacement, rotation and obliteration of the upper part of the pelvis by pressure. You could have said from that I think for sure that it was a retroperitoneal tumor probably of a pretty smooth contour, and probably not outside the kidney because a paranephritic abscess would not give that picture. An appendiceal abscess surely would not give a picture of that type. The question of the condition of the patient enters there and it is a matter of judgment whether the surgeon considers it safe to have cystoscopy and pyelography done.

Dr. H. B. Sweetser (closing): The patient's poor condition precluded any attempts at refinement of diagnosis. The general symptoms, with the high leukocyte and the large tender swelling, clearly demanded exploration as an emergency operation, and there could be no mistake as to the site of attack. The diagnosis was not evident till after the tumor was removed.

PERNICIOUS ANEMIA IN A BOY AGED ELEVEN

REPORT OF A CASE

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and
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The response of pernicious anemia to liver and liver extract therapy is now recognized as being so characteristic that it has practically become one of the postulates to which the diagnosis of this condition must conform before it is accepted as being correct. This has recently been pointed out by Faber[1] in a report in which he has used such response as a means of identification and classification of severe pernicious-like anemias in a small series of infants and children. In this report he concludes that one of his cases, aged nine and a half months, showing the blood picture indicative of pernicious anemia, and having achlorhydria

[1]Faber, Harold K.: The value of liver extract (343) in identifying and in treating certain anemias of infancy and childhood. Am. Jour. of Dis. of Chil., 36:1121 (December) 1928.

and the characteristic response to liver extract therapy, is the youngest authentic case of pernicious anemia on record, provided that these criteria are acceptable as conclusive.

Prior to the introduction of liver in the treatment of these cases, a fair number of severe pernicious-like anemias have been reported as occurring in early childhood. In his report, Faber, reviewing these previously reported cases, calls attention to the fact that the diagnosis has been dependent entirely upon the clinical blood examination during life and the postmortem study.

It must be admitted, however, that pathologic examination has quite failed to offer satisfactory and substantiating proofs of diagnosis of pernicious anemia. In fact, the advent of liver therapy has brought about a radical change in the very clinical-pathologic conception of this disease. It is now generally conceded that the process is not an over-destruction of red blood cell elements, but rather some factor, probably associated with the liver, which retards and inhibits normal erythroblastic maturation.

Assuming this concept to be correct, one may then reason that the excess pigment found in the blood, skin, liver, duodenal contents, etc., are there, not because of over-destruction of blood, but simply because the supply of hemoglobin pigment in the body exceeds the demand in the bone marrow, in turn due to retarded liberation of blood cells. The over-mature, extreme right shift neutrophils with thinned out, stranded, and over-segmented nuclei have been described chiefly by Watkins[2] as being characteristically pernicious anemia neutrophils. It is his opinion that the presence of such neutrophils is diagnostic of pernicious anemia and that these morphologic characteristics are the result of this same process of prolonged maturation within the bone marrow.

The following case report is presented with the desire to illustrate several interesting features:

1. Pernicious anemia occurring at the age of eleven years is sufficiently rare to entitle it to special attention.

2. The comparatively greater accuracy (and withal greater satisfaction) that the therapeutic test of response to liver extract offers in place of necropsy criteria; and

3. To illustrate an unusually marked reversion of the neutrophils to the type described by Watkins as characteristic and diagnostic of pernicious anemia.

CASE REPORT

P. K., aged 11, was admitted to St. Mary's Hospital, Dec. 21, 1928, having been referred by Dr. Dan Clark of Pine City, Minnesota.

Present Illness.—On or about the first of December the patient contracted a cold. This condition "developed into an influenza," from which he seemed to be convalescing favorably until a day or two before admission. At this time the chief symptoms were pyrexia, extreme exhaustion, and shortness of breath.

Family History.—The father, mother, one brother and one sister are living and well. The father's hair turned gray in the early twenties, and, on questioning, he states that he has noticed prickling and shooting pains through his tongue, off and on, for two or three years. His blood picture, however, was entirely normal and gastric analysis showed normal acidity. Likewise, the mother's blood picture and gastric analysis were normal.

Past History.—The patient was said to have been fairly healthy as an infant and escaped the usual chil-

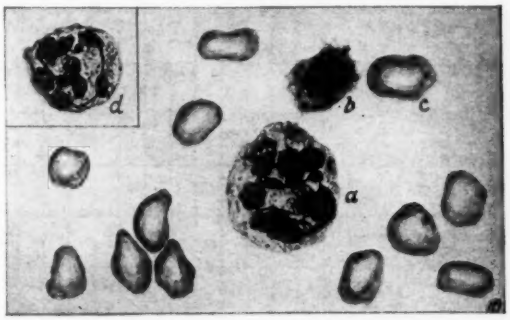

Fig. 1. *a*. (128.4)-(64.2) and *d*. (130.3)-(62.3) P. A. neutrophiles. *b*. (127.4)-(65.4) nucleated red blood cell. *c*. Erythrocyte.

dren's diseases, except for an attack of scarlet fever which occurred at the age of seven. Tonsillectomy was performed in October, 1927. The parents observed that the child had seemed unusually pale and weak for about one year prior to the present illness.

Examination.—In general, the child was undernourished, markedly dehydrated and of small stature. He weighed fifty-four pounds. On admission to the hospital he appeared extremely pale and completely exhausted, almost to the point of being moribund. There was marked dyspnea and considerable cough. His temperature was 103.4. The throat was moderately inflamed and much mucopurulent nasal and post-nasal discharge was present. The ears were not suppurating, but the drums were injected. The mouth was dry and much sordes appeared on the teeth and tongue. The cervical and submaxillary glands were distinctly enlarged. Examination of the lungs was negative except for occasional bronchitic râles. The heart rate was very rapid. A hemic murmur could be heard over the precordium, but there was no cardiac enlargement. At first there was much distention of the abdomen, but later a moderately enlarged spleen could be palpated. The extremities were negative and no abnormal reflexes were noted.

Laboratory Data.—Urinalysis: alkaline reaction; no albumin or sugar; only occasional leukocytes and epithelial cells present on microscopic examination. Blood: Hemoglobin (Sahli), 25 per cent. Red blood cells, 1,450,000. White blood cells, 20,100. Of the latter, 70 per cent were polymorphonuclears. The morphology of the cells on smear indicated pernicious anemia. Anisocytosis and poikilocytosis were marked. There were numerous macrocytes, many of which were polychromatophilic or loaded with basophilic granules. The cells were generally well filled with hemoglobin. A

[2]Watkins, C. H. "A classification of chronic idiopathic secondary anemia, with especial reference to the morphology of the blood." Jour. Am. Med. Assn., 93:1365, Nov. 2, 1929.

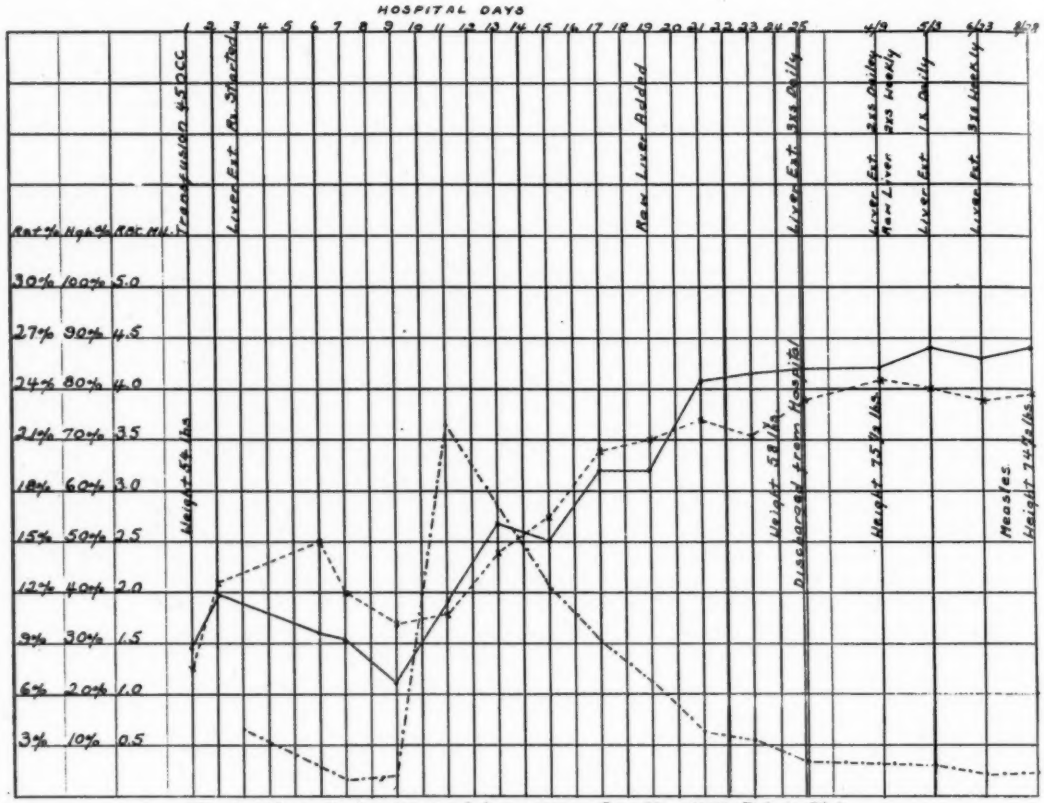
HOSPITAL DAYS
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 4/9 5/13 6/23 8/29
Transfusion 450 cc
Liver Ext. Ps. Shot Daily
Raw Liver Added
Liver Ext. 3xs Daily
Liver Ext. 3xs Daily
Raw Liver 3xs Weekly
Liver Ext. 1x Daily
Liver Ext. 3xs Weekly
Height 54 lbs.
Height 58 lbs.
Discharged from Hospital
Height 75 1/2 lbs.
Measles
Height 74 1/2 lbs.
Red% Hgb% Red Mil.
30% 100% 5.0
27% 90% 4.5
24% 80% 4.0
21% 70% 3.5
18% 60% 3.0
15% 50% 2.5
12% 40% 2.0
9% 30% 1.5
6% 20% 1.0
3% 10% 0.5

Fig. 2. Chart showing hematopoietic response. Case No. 63707, Duluth Clinic.
——— hemoglobin.
——— erythrocyte count.
.—.—.—. reticulocytes.

few normoblasts and an occasional megaloblast were noted. Many of the polymorphonuclears were rather larger than normal and showed very definite oversegmentation and stranding. Some of these nuclei contained as many as eight to ten thinned out lobules* (Fig. 1).

Progress.—Owing to the patient's precarious condition, he was given a transfusion of 450 c.c. of citrated blood on the day of admission. The next morning there was noted a purulent discharge from the left ear. The patient's condition seemed definitely better than on the preceding night. The blood examination following the transfusion indicated a hemoglobin of 42 per cent (Sahli), red blood cells 1,908,000, and white blood cells 12,700.

In the meantime, further evidence and laboratory data were obtained as follows: Stools were negative to occult blood; no evidence of ova or parasites. Blood culture showed no growth. Blood Wassermann returned negative. Fragility test showed beginning hemolysis at 0.42 and complete hemolysis at 0.32. Clot retraction reported as very slight. Aspiration of the stomach after test meal was attempted about a week after admission, but only a very small amount of material could be obtained; in fact, there was some doubt as to whether or not it was actually stomach contents. This showed no free hydrochloric acid and only a weak qualitative reaction of combined acids. Examination of the vomitus on the following day, however, showed no free hydrochloric present and a combined acidity of 26.

On Dec. 25, 1928 (fourth hospital day), treatment with liver extract was begun, using four vials (Lilly's 343) daily. A few days later the dosage was increased to six vials with the addition of a raw liver cocktail once daily. The diet was as general as possible and included certain portions of meat, fruit and cooked vegetables each day. On Dec. 28, 1928, the right ear drum showed injection and bulging. Myringotomy was done with free purulent discharge now coming from both ears.

The hematopoietic response to the above treatment can best be appreciated by noting the accompanying chart (Fig. 2) with attention to:

(1) The transient response to the blood transfusion.

*A later slide was examined by Dr. Charles Watkins of the Mayo Clinic in which he noted one neutrophil having 14 distinct lobulations. He stated in a personal communication that the morphology of these cells was diagnostic of pernicious anemia and that the smear represented the most extreme instance of right shift neutrophils that he had ever seen.

(2) The sharp sudden rise in the reticulocyte count occurring on the seventh day of liver treatment and the gradual decrease coincident with the rise in the hemoglobin and the red blood cell count towards normal.

(3) The somewhat late reticulocyte response (probably the transfusion and the severe infection were factors in delaying this).

Coincident with the clearing up of the infection the temperature gradually dropped to normal, and the child's appearance improved accordingly. Before any appreciable effect was noted in the blood counts themselves, a very obvious change in the patient's general attitude, appearance and appetite became noticeable very soon after beginning the administration of liver extract. This improvement continued, and at the time of the patient's discharge from the hospital on the twenty-fifth day, he was able to be up and about with ease and comfort.

After leaving the hospital the patient was observed from time to time and continued to gain in weight and strength. On March 21, 1929 (three months after admission to the hospital), it will be noticed from the chart that his count was remaining quite normal. His weight at that time was 75.5 pounds, a gain of 20 pounds since admission. The parents stated that now for the first time in his whole life was he able to cope with other boys of his own age.

Of further interest was the fact that another opportunity was afforded us to determine the patient's gastric acidity. At this time aspirations were made at five minute intervals following the injection of one-third mgm. of histamin, with the result that free hydrochloric acid was found to be present at the end of each period. The free acid values were low, however, specifically, six, five and q.n.s. (but faintly present on qualitative test).

Conclusion and Comments.—The case reported is considered to be a true pernicious anemia occurring in a boy eleven years of age in which the usual clinical feature indicative of pernicious anemia (with the exception of absolute achlorhydria) was further corroborated by the presence of unusually well-marked characteristics of the so-called "pernicious anemia neutrophil" and by the typical hematopoietic response, as well as the general constitutional reaction, to liver therapy.

One may well ask the question whether we as yet know that we have a fixed syndrome to which we may continue to give the name "pernicious anemia," and exclude from it border-line conditions which closely approximate its major findings. Quite likely the whole story has not yet unfolded itself despite the widespread interest stimulated by the results of liver therapy. The observations on this boy were carefully made and it is hoped that similar studies in young anemics may throw more light, not only on the hematology involved, but on the genesis of the all-important achylia—so striking an accompaniment in adult cases.

RADIOACTIVE WATERS AND SOLUTIONS

Not many years have passed since the Council on Pharmacy and Chemistry, basing its decision on the then available evidence, admitted to New and Non-official Remedies various preparations containing in solution radium or radium emanation (radon), and various devices for causing radium emanation to pass into drinking water. The evidence was not extremely well controlled or profuse in amount, but there seemed to be a demand by physicians for such preparations and the Council considered it worth while to set up at least minimum standards of radium content or radium activity. Actually, innumerable preparations were on the market which contained insufficient radium to have any demonstrable effects. Now the Council has issued the following statement: From an examination of the available evidence, it appears that the value of the internal use of radium solutions or of water containing radon in chronic arthritis, gout, neuritis and high blood pressure is not demonstrated by controlled clinical evidence; that, in spite of many years of trial, acceptable evidence has not become available and until such evidence does become available the Council has decided not to accept generators for the production of water charged with radon or radium solutions intended for intravenous use. The announcement by the Council disposes of the claims made for all sorts of solutions and for the devices to be used in preparation of such solutions, whether they contain considerable amounts of radium or but insignificant traces. (Jour. A. M. A., September 7, 1929, p. 771.)

LIVER EXTRACT NO. 343

The Council publishes a report of the Committee on Pernicious Anemia of the Harvard Medical School. This report states that in May, 1927, the Committee on Pernicious Anemia of the Harvard Medical School was organized to study the properties and to determine the clinical value of the fractions of liver that were being extracted, and to determine in what way a satisfactory product could be made available. Under direction of this Committee, Eli Lilly & Co. offered to manufacture one of the extracts developed. The function of the Committee was merely to supervise the production of a suitable extract of known potency until such time as the medical profession should have become accustomed to its use. The treatment of more than 100 cases of pernicious anemia with this extract indicated that a satisfactory product was available and it was accepted by the Council on Pharmacy and Chemistry for New and Non-official Remedies, under the name "Liver Extract No. 343." For the past year Eli Lilly & Co. has regularly produced lots of material every one of which has been shown to be clinically effective in the treatment of pernicious anemia, by a standardized process approved by the committee. The committee now feels that its function of developing a reliable commercial product has been accomplished, and that it may therefore cease actively to supervise the manufacturing process. (Jour. A. M. A., October 12, 1929, p. 1144.)

PRESIDENT'S LETTER

VALE

AS the year 1929 enters upon its closing month your president feels he must needs give expression of appreciation for the whole-heartedness of the counsel, the support given him by the officers and the various committees, and of the good will as well as the many kindly expressions bestowed by the membership of the State Society.

No one can have any doubt of the future progress of the Minnesota State Medical Association. The year has been one of accomplishment but much remains to be done if we are to maintain our logical position as leaders and educators of public opinion in matters pertaining to the health and welfare of this commonwealth. The affairs of the Association are in good and competent hands, men whose minds are attuned to future progress and whose sense of duty is in accord with the spirit of the times. To them we commit the furtherance of projects under way and others as they may arise, knowing full well that they will "carry on." To all of you our grateful thanks are extended, coupled with the hope that each of you may continue to enjoy that prosperity your efforts so richly deserve and that you and yours may experience a joyous Christmastide and a glad New Year.

J. T. Christison

President,
Minnesota State Medical Association.

EDITORIAL

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Vol. XII December, 1929 No. 12

THE COST OF MEDICAL CARE

Previous mention has been made editorially* of the investigation being made by the Committee on the Cost of Medical Care. This representative committee headed by Dr. Ray Lyman Wilbur, for a number of years president of the Leland Stanford Jr. University and now Secretary of the Interior, has obtained the services of several statisticians, sociologists and economists, in addition to utilizing statistics already gathered by numerous public health agencies, insurance companies and the like, in order to obtain accurate information bearing on the many phases of the subject being investigated.

The undertaking is to take five years. Now after some two years of investigation abstracts of the first three publications are available. As the general subject of medical economics is of such lively interest to the profession it is our purpose to give editorial mention to these publications from time to time.

The purpose and scope of the investigation form the subject of the first abstract published. This was covered in our previous editorial.

The second abstract deals with the extent of illness prevailing in the United States. The figures—some accurate and some admittedly estimates—are doubtless as reliable as it is possible to obtain. Many of us have known in a general way much of the material presented but it seems worth while to report some of the figures gathered by the committee.

We are informed that on the average men are disabled by illness once a year for a period of seven or eight days; women once to twice a year for eight to twelve days; and children more than twice during the school year.

Respiratory diseases such as colds and grippe are far ahead of other groups of disabling diseases. Digestive disorders, throat infections, non-venereal diseases of the genito-urinary tract, dysmenorrhea and skin troubles all are important from a disablement standpoint.

Chronic diseases which are particularly burdensome from a financial standpoint were headed, according to a survey made in Boston in 1927, by heart disease, organic nervous and mental diseases, cancer and arthritis. Over 10 per cent of these cases had lasted ten years or longer at the time of the survey.

The prevalence of disease in the country which is distinctly preventable is appalling. Venereal diseases account for one person in every one hundred being under the care of a physician at any one time. Approximately

*The Five Year Program of the Committee on the Cost of Medical Care. MINNESOTA MEDICINE, August, 1928, XI, 553.

700,000 persons in the country are afflicted with tuberculosis at all times. A million cases of malaria exist each year. Thirty-six thousand cases of smallpox were reported in a recent year. It is estimated there are 110,000 narcotic drug addicts. The complications incident to the two million births in the country each year, which are higher in percentage than in most civilized countries, are preventable in a large degree. The hospitalized mental cases number 350,000. This number equals the total number of available hospital beds for all other conditions and does not include the total number of mental defectives in the country, which is estimated at 900,000 (feeble-minded, imbeciles and idiots).

Preventive measures should be able to cut down this array to a large extent. The stupendous number of mental cases, a large portion of them hereditary, constitutes a eugenic problem which organized society has almost entirely ignored and eventually will have to face.

The medical facilities in this country is the subject of the third abstract published. It is estimated that there are some 1,500,000 persons employed in connection with the care and prevention of illness. There are more physicians in this country for the population than in any other country in the world. The number per 100,000 population varies from 71 in South Carolina and Montana to 200 in California.

Of the 143,000 practicing physicians, 19,000 limit their practice to a special field, 20,000 practice a specialty but do not limit themselves to it. Those on a full time salary basis number about 11 per cent of the total. Dentists number 67,000; registered nurses, 200,000; practical nurses, 152,000; midwives (chiefly in the southeastern states) 55,000; irregular practitioners (osteopaths, chiropractors, Christian Scientists, etc.), 32,000.

A consideration of the hospital situation reveals that the number of available beds has doubled in the past twenty years. Still, about 40 per cent of the counties in the United States have no hospitals for general community use. Considerably more than half of the entire hospital bed capacity is provided by federal, state and local governments. More than one quarter of the beds are provided by non-profit organizations for public service, controlled by independent boards of trustees, churches and other bodies.

The share that hospitals take in the cost of medical care has received the focus of public attention recently. Generalities on the subject are dangerous. With a doubling of available hospital beds it would not be surprising if certain localities found themselves overhospitalized and the undesirable necessity of competition between hospitals arose. We venture to predict, however, that investigations which we understand are being carried out will show that the public gets more from their expenditures for hospital service than they get from hotels, for instance, which of course is as it should be, since most hospitals are tax-free, have received donations to a greater or less degree, and are not operated for profit.

We all have our own ideas regarding the cost of medical care. When we consider the increase in the nation's expenditure in recent years for such out-and-out luxuries as cosmetics, tobacco and alcohol and such debatable necessities as automobile and radio, it is evident that the wherewithal is not lacking but the problem is primarily one of distribution of expenditures with some regard to relative importance of things. This will doubtless never be completely accomplished in a country where the income of prize fighters and movie actors is measured in the hundred thousands, absolutely out of proportion to the character of services rendered.

Education of the public should accomplish something and if the Committee on the Cost of Medical Care after its study has no specific recommendations to offer for reducing the actual cost of medical care but merely demonstrates that the various medical commodities are not relatively too expensive, the investigation will have been worth while.

INTRAVENOUS ANESTHESIA

To observe the development within a few minutes of deep sleep on the administration of intravenous medication is striking to say the least. The use of sodium iso-amyl ethyl barbituric acid, otherwise known as amytal, promises to be an addition in the field of anesthesia. How fully it will replace the inhalation anesthesia, however, is questionable.

The replacement of cholorform by ether was a distinct advance in the field of general anesthesia from the standpoint of safety. Ethylene gas has

distinct advantages over ether in certain cases. Intraspinal anesthesia has had a distinct revival in recent years although its field is necessarily limited. There will always be a large field for local anesthesia. In spite of the boon that anesthetics of all kinds have been to the human species, the last word on the subject of anesthesia has not been said and efforts to improve present methods will continue.

The barbituric acid derivatives of late have come into widespread use as hypnotics. The hypnotic action of amytal given intravenously is its outstanding feature. This action may be augmented by the previous administration of morphine or chloretone or both. In many instances, however, this hypnotic action is not sufficient to permit major surgical procedures without the assistance of slight amounts of other anesthetics.

In many operative cases, however, there is a distinct advantage in being able to put the patient to sleep either in bed or in the operating room preceding the administration of other anesthetics. The psychic effect of the surroundings preceding an operation are not to be ignored. The prolonged quieting effect which persists as long as a day or two following the giving of amytal intravenously has advantages but it also has distinct disadvantages. Too much quiet following operation increases the likelihood of postoperative pneumonia or massive lung collapse. Doubtless, this tendency could be overcome by carbon dioxide and oxygen inhalations. Then, too, experience has shown that postoperatively these patients require constant watching because of an excitation stage which sometimes appears as the effect of the drug diminishes.

The intravenous administration of a drug to produce general anesthesia is necessarily fraught with increased danger. In case of excessive action due to overdose or idiosyncrasy the anesthesia cannot be interrupted as with the inhalation method. The rectal administration of amytal which has been used with some success would permit at least the removal of the drug from the rectum if evidence of overaction arose. On the other hand rectal medication of any sort is uncertain.

It seems premature even for those who have had most experience with amytal administration intravenously to predict the place this anesthesia will ultimately take as a general anesthetic. That it will have its special field seems likely.

A PHYSICIANS' SERVICE BUREAU

Not so many years ago it was not unusual for the general practitioner to send out his statements only once or twice a year. If his fees were paid—all right; if not—all right. Those which were not paid were crossed off the books and considered bad debts. Such were in effect charity cases. The medical profession rightly earned the reputation of being poor business men.

Times have changed. It is now usual for the physician to send out his statements the first of each month. The adoption of modern business methods by the profession has been accepted. In the larger communities hospitals and clinics are maintained for those unable to pay. Here the physician donates his services in the great majority of instances without financial compensation. In addition to this free service most physicians have a certain number of charity patients to whom for one reason or another they give their service gratis.

With the organization of charity medical service, however, the physician must count on his private practice to furnish the wherewithal to keep the wheels going. It seems oftentimes that doctors' bills are among the last to be paid. The family budget, if there is one, provides for current expenses and purchases on the instalment plan but as a rule no provision is made for such emergencies as accident or sickness. Physicians have found it necessary to obtain assistance in collecting unpaid accounts by employing collectors. The methods used by collection agencies as a whole may be perfectly correct but how many physicians know just what methods are used in bringing pressure to bear on their poor accounts?

In addition to bad accounts the physician has a variety of financial interests which can be handled better by group action than by individuals. The physicians' telephone exchange has come apparently to stay. This activity is less likely to be abused when controlled by a representative committee of physicians than by a lay supervisor. The telephone companies have a way of arbitrarily fixing rates for classified directory insertions and extra insertions where group action might prove more effective in preventing unfair rates. A physicians' service bureau can be of material assistance in procuring trained or practical nurses, blood donors, and so forth.

There are just enough deadbeats in every community who go from physician to physician with no intention of ever paying their bills to warrant their classification from a credit standpoint.

These are the activities which have been undertaken by the Physicians' and Dentists' Service Bureau of Saint Paul. Launched July 1, 1928, under the supervision of Mr. H. M. Temple, Jr., the membership has grown from 183 to 250 in a year's time and promises to increase steadily as time goes on. After a year of operation the Bureau has published a confidential guide containing the names of cases which have been referred to the Bureau and ratings in accordance with the manner in which the accounts have been taken care of.

As a confirmation of the idea that physicians should have supervision of the collection of poor accounts, the Bureau has unearthed several instances where it has been discovered that certain accounts had been paid previously to employed collectors but the individual's name had been returned to the physician as an uncollectable account.

A further activity recently added to the Bureau is the handling of instalment payments to physicians. While a large percentage of physicians' accounts are paid in instalments—in many cases very irregularly—it is felt that the slight increase in cost to the physician incident to handling by the Bureau will be more than compensated by more regular payments when made directly to the Bureau. No attempt is being made so far to actually make any cash loans to patients and immediate cash payments to physicians according to the method recently proposed in Chicago which attracted considerable publicity.

Physicians in Saint Paul are not the first to form such a bureau. In many smaller communities physicians have been in the habit of allying themselves with merchants and other professions for mutual protection. Some nineteen cities already have established physicians' bureaus, which list includes the cities of Atlanta, Louisville, Richmond and Jackson (Mississippi). The Saint Paul bureau is already reciprocating with similar bureaus in Memphis, Portland (Oregon), Mineola (New York), and Lexington (Kentucky). The idea is a good one and indications are that new bureaus will be established in the larger centers of population all over the country.

HISTORICAL COMMITTEE

In these days of laboratory and machine-made diagnosis and treatment it is difficult to realize how the generations preceding us practised medicine. Diagnosis and treatment was an art then. Reading the old works shows much keen clinical observation that is often lacking nowadays. Who knows who brought the first stethoscope to Minnesota? We know one was brought here in 1859. Who can substantiate an earlier date? The clinical thermometer was used in St. Paul for the first time about 1870, and we know who introduced it. Do you realize that the hypodermic syringe was not used in the United States till 1856, and that the use of the clinical thermometer and microscope is not mentioned in the Harvard Medical College Catalogue till 1867-1868. A Minnesota physician in a case report published in 1856 states that microscopic examination of a tumor of the stomach showed cancer cells. Can you inform your Historical Committee regarding the use of these instruments in the early days in Minnesota? If so, communicate with Dr. H. M. Workman at Tracy.

J. M. A.

THE ANTIPELLAGRIC VITAMIN

Evidence has been furnished that the so-called accessory food factor formerly designated as vitamin B and supplied in comparative abundance by yeast apparently contains, in addition to the antineuritic vitamin, a factor which promotes growth and cures and prevents dermatitis in rats; consequently it has been regarded as identical with the "P-P" factor described by Goldberger and others as curative and preventive of human pellagra. The newest American designation of this is vitamin G—the vitamin B_2 of British biochemists. There is little doubt that both of these water-soluble vitamins are essential to growth and well-being; and it seems reasonably certain that pellagra is due to a vitamin deficiency. It is now known that unheated yeast is rich in both and that cereals contain more vitamin B than vitamin G; milk and meat, the reverse. The vitamin G value of wheat and maize is low, as is that of dried legumes such as peas. Meat and egg yolk are richer in vitamin G than are the cereals, while liver and fresh milk are excellent sources of this dietary adjuvant. (Jour. A. M. A., October 12, 1929, p. 1149.)

OBITUARY

Louis A. Nippert

1860-1929

Dr. Louis A Nippert, one of the most widely known internists of Minneapolis, was fatally injured when he fell from his horse as it jumped from the path of an oncoming automobile, the evening of November 6, 1929. Dr. Nippert had a wide acquaintance in Minneapolis, having practised there for forty years, and was known and admired by the many graduates of the University medical school through his medical clinics given over a long period of years.

Dr. Nippert was born in Basle, Switzerland, July 14, 1860, of American parents. His father, the Reverend Louis Nippert, was in charge of Methodist missions in Switzerland. Dr. Nippert came to the United States when seventeen years old and lived for some years in Cincinnati, where he graduated from the Miami Medical College of Cincinnati University in 1883. He then spent two years studying in Paris and Vienna before beginning practice in Minneapolis.

For many years Dr. Nippert was on the faculty of the medical school at the University of Minnesota. He had been on the staff of Hillcrest Hospital since its organization. In 1898 he was president of the Hennepin County Medical Society, and in 1912, president of the Minnesota Academy of Medicine. He was a member of the Nu Sigma Nu medical fraternity and also of the Minneapolis Club.

Dr. Nippert is survived by his wife; a daughter, Mrs. Edgar Zelle, of Minneapolis; four brothers, Dr. Henry T. Nippert of Saint Paul, Dr. Edgar Nippert of Los Angeles, Judge Alfred Nippert and Herman Nippert of Cincinnati; and three sisters, Mrs. Helminge of Seattle and the Misses Marion and Eleanor Nippert of Cincinnati.

While Dr. Nippert has not been in general good health for several years, his sudden death came as a shock to his many friends. His upright character and his scientific attainments endeared him to all who knew him.

William J. Byrnes

1859-1929

Dr. William J. Byrnes, a prominent northwest physician and surgeon and former Hennepin county coroner, died suddenly Monday morning, Nov. 4, 1929, at his home, following a heart attack. He was 70 years old.

Dr. Byrnes was active until the time of his death and for many years maintained offices in the Masonic Temple building.

He was born in Minneapolis and received his early education in the schools of that city. As a youth he attended St. John's university at Collegeville, Minn. Later he attended the University of Michigan Medical school, and following his graduation in 1882 he went abroad to study. After practicing in Minneapolis for several years he was named coroner.

In 1889 Dr. Byrnes was elected president of the Hennepin County Medical Society, serving a one-year term. In 1927 he was elected an honorary member of the society.

During the World War, despite the fact that he was too old for active service, Dr. Byrnes carried on volunteer medical work among service men in his home city. He was active in work of the Civic and Commerce Association and was also a member of the Elks and Royal Arcanum lodges.

Surviving Dr. Byrnes are his wife; a son, Dr. W. A. Byrnes of San Diego; three daughters, Lyle Brynes, Mrs. Hallam Huffman of Chicago, and Mrs. Robert J. Seiberlich of Minneapolis.

Mary Whetstone

1849-1929

Dr. Mary Whetstone, 80 years old, the second woman to practice medicine in Minneapolis, and a resident of the city for 40 years, died Thursday, Oct. 24, 1929, at the Minnesota Soldiers' home.

Dr. Whetstone was a graduate of the University of Michigan, having received her medical degree from that institution in 1882. She attended clinics in Detroit, Boston, New York, London, Paris and Vienna. In 1882 she married Dr. Allan S. Whetstone.

She was instrumental in establishing Northwestern Hospital of Minneapolis and in securing its present site. Dr. Whetstone was the first woman to be elected vice president of the State Medical Association, and the first woman physician appointed on the staff of Asbury and City hospitals.

Besides being prominent in medical circles, Dr. Whetstone was active in various women's organizations, having served as superintendent of medical temperance for the W. C. T. U. and as a promoter of the Minneapolis Mycological Society. She is credited with being responsible for introducing the teaching of cooking in the public schools in Minneapolis.

She was an active worker for woman suffrage.

Surviving are two sisters, Mrs. Margaret Wimer of St. Louis, Mo., and Mrs. Sumner Lord of Marshalltown, Iowa.

A PAGE FORUM OF THE COMMITTEE ON PUBLIC HEALTH EDUCATION

These views on state medicine from the *Chicago Journal of Commerce* and the *Illinois Medical Journal* will be of interest:

"President Frank of the University of Wisconsin has declared in a speech before the American College of Surgeons that unless medical practitioners "excel the industries, insurance companies, and governments in their zeal for the promotion of preventive medicine," the present individualistic medical profession is certain to be "superseded by state medicine or its equivalent." This is a loose use of words, since the possible equivalent which he foresees is not really an equivalent; it is "a vast high-powered medical machine, under the sponsorship of industries, insurance companies and governments." A private medical system supervised by governmental agencies is not the equivalent of a governmental system. It means bureaucracy, not socialism. But either of these alternatives would destroy the individualistic medical profession.

"It has been evident for some time that the profession cannot survive in its present form. It does no good for the doctors to attribute the high cost of sickness to hospital and laboratory charges, and to the demands of patients for private rooms and private nurses, whether they need them or not. The plain fact is that the doctors get little sympathy from the public.

"The public is sympathetic to a revolution in the economics of medicine. Unless the profession itself evolves methods for reducing the cost of sickness, corporation medicine will probably become dominant within the next generation."—*The Chicago Journal of Commerce.*

"State operation and state control of medicine ruined medical practice in Germany and has given the people of that country the worst medical service administered in any civilized country in the world.

"When an institution such as a university or a corporation enters into the practice of medicine, as is the case in many instances at the present time, there come up certain points of interest to the medical profession.

"A university is endowed for education, research and scholarship, and when it enters the practice of medicine it is dealing unfairly with the private practitioner. 'Unfair,' because this institution has the enormous advantage of reputation and size to attract patients. 'Unfair,' because even if it charges large fees its prestige would make it an impossible competitor with the private practitioner.

"Much is being said by economists about only the very wealthy and the very poor getting the maximum of medical service, and so leaving the great middle class unable to procure the so-called maximum service. This statement is not accepted by thinking medical men. We believe that the private practitioner is giving good service and sees that his patients get what they need at a price they can afford to pay.

"There is a strong tendency at the present time to systematize or machine out the most desirable thing in the practice of medicine, and that is, the very personal contact of physician and patient. This personal contact cannot be removed without jeopardizing the best interest of the physician, the patient and the public. Chain store or department store medicine is detrimental to the welfare of both the physician and the public.

"It is important under the present standard of ethical procedure that groups practising medicine must adhere to the same ethical principles as regulate individuals. Because of their size and influence corporations are in no way relieved of the ethical responsibility that applies to general practitioners. If the staff of corporations engaged in the practice of medicine fails to recognize this fundamental principle then all the men connected with the staff should be amenable to the same discipline as if they were individual practitioners."—*Illinois Medical Journal.*

OF GENERAL INTEREST

Dr. O. E. Locken was re-elected mayor of Crookston at the biennial municipal election held in November.

Dr. and Mrs. Adam M. Smith have returned from Europe, where Dr. Smith was doing post-graduate work in Vienna. He will limit his practice to internal medicine.

Dr. W. K. Foster, for a number of years instructor of gymnastics at the University of Minnesota, has resigned his position to enter private medical practice in Minneapolis.

Dr. W. Andrew Bunten of Rochester has announced the opening of offices at Suite 654 Fisher Building, Detroit, Michigan, practice limited to neurosurgery and neurosurgical diagnosis.

On October 12 Dr. Raymond Sorenson severed his connections with Dr. W. R. Humphrey at Stillwater, Minnesota, to accept a position with the U. S. Government Hospital at the National Soldiers' Home, Danville, Illinois.

The marriage of Miss Dorothy Dean and Dr. Louis Good took place Thursday, October 31, in Saint Paul. Dr. Good will enter the practice of surgery in Texarkana, Texas, with offices in the Texarkana National Bank Building.

The Mississippi Valley Dermatological Conference was held at the University Hospital on Friday, October 25, and at the Mayo Clinic on Saturday, October 26. There were fifty-three visitors from Chicago, Cincinnati, Milwaukee and St. Louis.

The marriage of Miss Margaret Wirt Ritchie of Little Falls, Minnesota, to Dr. Asher Abbott White, son of Dr. and Mrs. S. Marx White of Minneapolis, will take place early this month, according to a recent announcement of the engagement.

Dr. C. H. Sherman of Bayport, Minnesota, is attending the Graduate School of the University of Pennsylvania, where he is working for a Master's degree in surgery. Dr. E. V. Strand, a recent graduate of the University of Minnesota, has taken over Dr. Sherman's practice at Bayport.

Dr. Howard McI. Morton of Minneapolis has returned from Holland, where he lectured before the International Ophthalmological Congress. Dr. Morton will make an intensive study of causes and treatments of blindness, as a member of an international committee on the prevention of blindness.

A report from the recent meeting of the American College of Surgeons gives a list of sixteen approved hospitals in Minneapolis, twelve in Saint Paul, with a considerable list scattered throughout other towns in the state of Minnesota. Dr. Malcolm T. McEachern, associate director of the college, stated at the meeting that the mortality rate in hospitals, now averaging 3.5 per cent, has been cut in half in the last decade. Twenty-three Minnesota surgeons were elected to fellowships at the meeting.

COMMUNICATIONS

Dr. E. A. Meyerding, Secretary,
Minnesota State Medical Association.

Dear Doctor:

I am in receipt of the Certificate of Honorary Membership in the Minnesota State Medical Association.

Owing to vacation time I am tardy in acknowledgment of this favor by my friends and associates in Minnesota, and ask you to convey to them my sincere appreciation of their kindness.

I miss the fellowship of the men I've worked with these years more than I can express.

I am glad to be able to do a limited work here. However, no place will be quite like Minnesota.

My best regards to Minnesota physicians, whom I shall enjoy seeing any time they pass this way.

Very sincerely yours,
C. A. DONALDSON, M.D.

To the Editor:

The following members of the Minnesota Medical Association were elected to Affiliate Fellowship in the American Medical Association at the Portland meeting:

Anderson, A. E., Minneapolis.
Bacon, Russell S., Montevideo, Minn.
Bell, John Wesley, Minneapolis.
Boehm, John C., St. Cloud, Minn.
Bole, R. S., St. Paul.
Bracken, H. M., Claremont, Calif.
Brown, E. J., Minneapolis.
Clark, Thomas C., Minneapolis.
Darling, J. B., St. Paul.
Davis, William, St. Paul.
Disen, Charles F., Minneapolis.
DuBois, J. A., Sauk Center, Minn.
Dutton, C. E., Minneapolis.
Haverfield, Addie R., Minneapolis.
Hawkins, V. J., St. Paul.
Hunt, F. N., Fairmont, Minn.
Johnson, H. C., St. Paul.
Johnson, H. P., Fairmont, Minn.
Linton, William B., Minneapolis.
Long, Jesse, Minneapolis.
Lum, C. E., Duluth, Minn.
Magie, William H., Duluth, Minn.
McComb, C. F., Duluth, Minn.
Mead, Marion A., Minneapolis.
Nippert, L. A., Minneapolis.
Richardson, W. J., Fairmont, Minn.
Schoch, J. L., New Ulm, Minn.
Simpson, J. D., Minneapolis.
Soderlind, A., Minneapolis.
Whitney, A. W., St. Paul.

Sincerely yours,
E. A. MEYERDING, M.D.,
Secretary, Minnesota State Medical Association.

CONSULTATION BUREAU

WM. A. O'BRIEN, M.D., *Director*
Minnesota State Medical Association
11 West Summit Avenue
Saint Paul, Minnesota

1. *Question.*—Patient came in complaining of vague abdominal pain. X-rays revealed no disease in the stomach, but mild chronic gall-bladder disease was present. Patient was put on routine medical treatment for one month with no relief. Cholecystectomy and appendectomy performed. Gall-bladder was apparently normal, slightly thickened wall, no discoloration, or increase in subserous fat layer; no stones found. Appendix showed adhesions, otherwise no change. Patient had normal recovery. Wound drained for two and a half weeks, last four days mostly serosanguineous fluid. Three weeks after operation, patient began to show jaundice. This started in region of incision, spreading upwards over arms and face, then down to lower extremities in next few days. Stools became clay-colored, urine dark, and skin itched. Jaundice became more marked in following weeks, until now, eight weeks postoperative, patient is 4 plus jaundiced. This jaundice is more marked in the morning than in the evening, after patient has been up and around. Since advent of jaundice, patient has been on fat-free, high-protein, low-carbohydrate diet, enteric coated magnesium sulphate pills, forced fluids, rest in bed, free catharsis and digitalis, minims 15 t.i.d.

Patient is well developed white female, age 57, with 4 plus jaundice all over body. Eyes normal. Sclera 4 plus jaundice. Mouth—no teeth. Tongue smooth at edges, normal size. Neck negative. Chest negative. Heart enlarged moderately to left, otherwise negative. Pulse rate 110, regular and full. Abdomen—no ascites present, tenderness in region of incision in right upper quadrant. Pelvic examination negative. Rectal examination—few internal hemorrhoids present, not large or bleeding. Extremities—slight swelling of ankles, no edema. Reflexes—nervous system negative throughout. No adenopathy.

Repeated urinalyses show bilirubin present, no albumin or sugar, 5-10 wbc, few hyalin casts per high power field (centrifugal specimen). Repeated blood studies show hemoglobin 80-86 per cent, red blood cells 4,000,000-4,600,000, white blood cells 6-8,000; differential count—polymorphonuclears 65, lymphocytes 28, eosinophils 2, mononuclears 5 (the highest reading on last test taken a week ago). No abnormal red or white cells.

The questions which arise are: What is the exact cause of the jaundice and how long is it advisable to wait before entering the abdomen again for exploration?

Answer.—The diagnostic possibilities are carcinoma of the head of the pancreas, cirrhosis of the liver, carcinoma of the biliary tract, stone in the common duct, and acute hepatitis of the liver. Liver abscess should be mentioned although the jaundice is usually not as severe as you have described. I would have mentioned acute catarrhal jaundice except for the fact that it has existed for such a long time.

We know of a similar experience in a patient. When first seen, the symptoms suggested acute catarrhal jaundice, but as time went on and the condition did not clear up, she was explored. The main complaint of the patient was toxic adenoma and the jaundice was only incidental. At operation, cirrhosis of the liver was found after jaundice of eight weeks duration.

There is only one concrete suggestion that we can offer. The patient should be explored as soon as possible. If the obstruction is malignant, an anastomosis between the duodenum and gall-bladder can not be made. If it is due to cirrhosis of the liver and the spleen is enlarged, a splenectomy is indicated. If a stone is present in the common duct, it can be removed; and if the condition is due to cholangeitis, the tract can be drained. I think you have waited a sufficient length of time before advising exploration. It is possible that the slight trouble in the gall-bladder may not have been responsible for the initial symptom.

2. *Question.*—In March, 1929, I operated on a woman, 50 years old, for a ruptured appendix and peritonitis. Six days later, it was necessary to put a tube in the bowel, which I did on the left side about the level of the umbilicus. She recovered and is in good health, but the fistula where I put the tube in the bowel failed to close. Her bowels move normally and the skin is in good condition. It drains about one or two teaspoons a day. Should I operate now to close it or wait longer? What operation is best? Please tell me what things to avoid.

Answer.—There is no great hurry in closing the fistula as long as the patient is not showing any signs of loss or irritation of the skin from the intestinal fluid. The second reason for delay is that these operations are often difficult and nature should be given a chance to help you.

Is the patient really suffering from an opening in the bowel? This may be determined by injecting barium or lipiodol into the sinus tract and taking *x*-ray pictures.

An attempt should be made to close the opening from the outside. If a piece of rubber tubing is pushed down the wound, it is sometimes possible to pull the base up against the opening. A rubber tube, 2 or 3 inches in length, is tied in the middle with a string. The tube is doubled back with the tied portion at the apex. This is pushed into the sinus tract, if the tract is large enough, and pulled tight against the opening if the tube has straightened out in the lumen of the bowel. A button may be placed over the opening and the tube tied with this. If healing occurs, all that is necessary to do is to cut the string and allow the tube to pass through.

If these methods fail, an operation may be indicated. The procedure may vary from a simple closure of the intestinal mucosa with inversion of the mucous membrane to a resection of the bowel with anastomosis. It is best not to try surgical treatment until other methods fail, and then be sure that you anticipate any difficulties that may arise.

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

AMERICAN COLLEGE OF PHYSICIANS, FOURTEENTH ANNUAL CLINICAL SESSION, MINNEAPOLIS, February 10-14, 1930

The American College of Physicians will hold its Fourteenth Annual Clinical Session in Minneapolis, February 10-14. Dr. John H. Musser, Professor of Medicine at the Tulane University School of Medicine, is President of the College this year. Dr. Sydney R. Miller, Associate Professor of Medicine at the University of Maryland School of Medicine, Baltimore, is President-Elect, and will be inducted to the Presidency toward the end of the Minneapolis Session. Dr. S. Marx White, Professor of Medicine at the University of Minnesota Medical School, is General Chairman of all Minneapolis Committees having charge of arrangements for the Clinical Session for the College in Minneapolis.

The Program provides hospital visits, clinics, demonstrations and ward-walks during the forenoons at various Minneapolis Hospitals; formal addresses for the general scientific sessions each afternoon and evening; a large medical exhibit and other features. All of the general assemblies, afternoons and evenings, will be held in the Minneapolis Auditorium. Eminent authorities in their special lines will present the results of their work before an audience competent to appreciate the value of the contributions.

The Convocation for the conferring of Fellowships will take place Wednesday evening, and will be followed by a reception and a program of general entertainment. Dr. John H. Musser, President, will deliver the chief address at the Convocation. The annual Banquet of the College will be held Thursday evening, February 13, at the Hotel Curtis.

Programs, details concerning reduced fares, admission of non-members, etc., may be secured from the Executive Secretary, E. R. Loveland, 133-135 S. 36th Street, Philadelphia, Pa.

MINNESOTA TRUDEAU MEDICAL SOCIETY

The annual meeting of the Minnesota Trudeau Medical Society was held at the Duluth Hotel, Duluth, Minnesota, at 6:30 P. M., October 19, 1929. There were twenty-eight members present.

The first business of the meeting consisted in a report from the various committees. Dr. Beadie of the Membership Committee submitted the following names for consideration of membership:

Dr. W. A. Colton, Veterans' Hospital, St. Paul.

Dr. L. H. Flancher, Sand Beach Sanatorium, Lake Park.

Dr. L. S. Jordan, Riverside Sanatorium, Granite Falls.

These members are to be acted upon at the next meeting.

Dr. D. G. Gardiner of Ancker Hospital, St. Paul, whose name was presented at the previous meeting, was elected to membership.

The remaining committees had no reports to make.

This being the annual meeting of the Society, a Nominating Committee was elected. On motion duly seconded, the following names were nominated and then unanimously elected for this committee:

Dr. F. L. Jennings, Glen Lake Sanatorium, Oak Terrace.

Dr. H. A. Burns, Minnesota State Sanatorium, Ah-Gwah-Ching.

Dr. S. A. Slater, Southwestern Minnesota Sanatorium, Worthington.

The following program was presented: "Experimental Tuberculosis by Intracerebral Inoculation" by Dr. Wm. H. Feldman of the Mayo Clinic, Rochester. Discussion followed by Drs. Laird, Hirschboeck and Lemon. "Forensic Medicine—Its Relation to Tuberculosis" by Mr. O. G. Haugland, attorney, secretary to the State Board of Law Examiners. Discussion by Drs. Tuohy and Harrington.

On motion duly seconded, the meeting adjourned.

ARNOLD S. ANDERSON, M.D.,
Secretary-Treasurer.

RICE COUNTY MEDICAL SOCIETY

A meeting of the Rice County Medical Society was held Friday, November 1, in the Stewart Hotel, Northfield, where a dinner was served under the auspices of Dr. P. A. Smith, of Faribault.

Dr. E. M. Hammes of St. Paul gave an interesting message on "Neurological Cases in General, with Special Reference to the Difficulties in Diagnosis."

Election of officers for 1930 resulted as follows:

President.....Dr. J. M. Murdoch, Faribault
First Vice President....Dr. Warren J. Wilson, Northfield
Second Vice President.....Dr. Edmund Backe, Kenyon
Secretary-Treasurer.....Dr. C. J. Plonske, Faribault
Delegate.....Dr. A. M. Hanson, Faribault
Censors—Dr. C. A. Traeger, Dr. W. H. Aumpf, Faribault; Dr. F. M. Babcock, Northfield.
Publicity Committee—Dr. W. H. Robilliard, Dr. S. B. Haessly, Faribault; Dr. F. M. Babcock, Northfield.

A vote of appreciation and thanks was extended to Dr. Hammes for his talk, to Dr. P. A. Smith for his hospitality and dinner and to Dr. A. M. Hanson for the use of his lantern machine.

C. J. PLONSKE, M.D.,
Secretary.

CAMP RELEASE DISTRICT MEDICAL SOCIETY

Officers elected at the annual meeting of the Camp Release Medical Society at Montevideo on October 14, 1929, are as follows:

President—Dr. M. A. Burns, Milan
Vice-President—Dr. J. S. Killbride, Canby
Secretary-Treasurer—Dr. L. J. Holmberg, Canby

Following the business meeting short addresses were made by Secretary Meyerding and President Christison of the State Association.

The Woman's Auxiliary held their annual meeting at the same time and elected officers:

President—Mrs. M. A. Passer, Olivia
1st Vice President—Mrs. H. M. Johnson, Dawson
2nd Vice President—Mrs. L. G. Erickson, Wood Lake
Treasurer—Mrs. L. G. Smith, Montevideo
Secretary—Mrs. L. C. Adams, Bird Island

At the evening meeting, following a joint dinner at the Riverside Hotel, Mr. F. Manley Brist, Attorney for the State Board of Medical Examiners, spoke on the work of his office. The address of the evening was a most interesting and illuminating presentation of the subject of anaphylaxis by Dr. Edgar T. Herrmann of St. Paul.

MINNESOTA ORTHOPEDIC CLUB

The semi-annual meeting of the Minnesota Orthopedic Club was held in Minneapolis November 1 and 2, 1929. There were ten members present.

A dinner meeting was held November 1, and clinical meeting at the Shriners Hospital November 2. Informal discussion and clinical material were presented to the members.

GEORGE A. WILLIAMSON, M.D.,
Secretary.

ST. LOUIS COUNTY MEDICAL SOCIETY

The following officers were elected at the Annual meeting of the St. Louis County Medical Society held October 10, 1929:

President.....Dr. F. J. Elias, Duluth
First Vice President.....Dr. C. W. Bray, Biwabik
Second Vice President.....Dr. O. E. Heimark, Duluth
Secretary-Treasurer.....Dr. M. McC. Fischer, Duluth

NEW AND NON-OFFICIAL REMEDIES

The following articles have been accepted by the Council on Pharmacy and Chemistry:

CUTTER LABORATORY
Diphtheria Toxoid-Cutter

ELI LILLY & CO.
Merthiolate

WINTHROP CHEMICAL CO., INC.
Luminal Capsules, 1½ grains

TRUTH ABOUT MEDICINES

Calcium Gluconate-Sandoz.—It contains calcium equivalent to not less than 12.40 or more than 12.80 per cent of calcium oxide. Calcium Gluconate-Sandoz is used to obtain the therapeutic effects of calcium. It is more palatable than calcium chloride and for hypodermic or intramuscular use is non-irritant. It is supplied in the form of a powder and in ampules containing 10 c.c. of a ten per cent stabilized supersaturated solution. Sandoz Chemical Works, Inc., New York.

Acne Bacillus Vaccine.—An acne bacillus vaccine (New and Non-official Remedies, 1929, p. 369) marketed in packages of one 5 c.c. vial, and in packages of one c.c. vial. Hollister-Stier Laboratories, Spokane, Wash.

Pertussis Bacillus Vaccine.—A pertussis bacillus vaccine (New and Non-official Remedies, 1929, p. 371) composed of several strains of pertussis bacilli, marketed in packages of one 5 c.c. vial, and in packages of one 20 c.c. vial. Hollister-Stier Laboratories, Spokane, Wash.

Staphylococcus Vaccine (Aureus and Albus).—A staphylococcus vaccine (New and Non-official Remedies, 1929, p. 375) prepared from *staphylococcus aureus* and *albus* in equal proportions, and marketed in packages of one 5 c.c. vial and in packages of one 20 c.c. ampule. Hollister-Stier Laboratories, Spokane, Wash.

Typhoid-Paratyphoid Vaccine (Prophylactic).—A typhoid vaccine (New and Non-official Remedies, 1929, p. 378) consisting of a suspension of killed typhoid, paratyphoid A, and paratyphoid B bacilli. It is marketed in packages of one 5 c.c. vial and in packages of one 20 c.c. vial. Hollister-Stier Laboratories, Spokane, Wash. (Jour. A. M. A., October 5, 1929, p. 1065.)

Atoquinol-Ciba.—The allyl ester of 2-phenyl-quinolin-4-carboxylic acid. The actions and uses of Atoquinol-Ciba are practically like those of cinchophen. It is supplied in the form of tablets, 0.25 Gm. (4 grains). Ciba Co., Inc., New York. (Jour. A. M .A., October 19, 1929, p. 1223.)

Chiniofon. — Sodium-iodoxyquinolinesulphonate.—A mixture prepared from approximately four parts of 7-iodo-8-hydroxy-quinoline-5-sulphonic acid, containing not less than 26.5 per cent of combined iodine, and 1 part of sodium bicarbonate. Chiniofon, which is closely similar to preparations introduced under various proprietary names as wound antiseptics, has been found to be of use in the treatment of amebic dysentery.

Bacillus Acidophilus Culture-Hollister-Stier. — A pure culture of *B. acidophilus* which contains not less than 150 million viable organisms *(B. acidophilus)* per c.c. at the time of issue. For a discussion of the actions, uses and dosage of bacillus acidophilus preparations see Lactic Acid-Producing Organisms and Preparations, New and Nonofficial Remedies, 1929, p. 220. Hollister-Stier Laboratories, Spokane, Wash. (Jour. A. M. A., October 26, 1929, p. 1309.)

PROCEEDINGS OF THE MINNESOTA ACADEMY OF MEDICINE

Meeting of October 9, 1929

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, October 9, 1929. Dinner was served at 7 p. m. and the meeting was called to order at 8 p. m. by the President, Dr. C. N. McCloud. There were 40 members and 1 visitor present.

Minutes of the September meeting were read and approved.

The scientific program consisted of the following:

DR. EMIL S. GEIST (Minneapolis) reported a case of "Internal Derangement of the Knee," with operation for removal of the foreign body. The patient was presented, and *x*-ray films were shown. He spoke as follows:

On October 3, 1928, I read a paper before the Southern Minnesota Medical Society entitled "Foreign Bodies in and Near Joints." This appeared a few months later in MINNESOTA MEDICINE. In this paper I reported twenty-two cases of joint disturbance due to the presence of a foreign body. In one-half of these the presence of this foreign body was unsuspected by the patient. The following case is another added to this list.

This patient, Mr. G. A. G. (Case No. 290200), was referred to us on March 18, 1929, by Dr. Linner of Minneapolis. He gave the following history:

The patient could not remember any injury whatever but said that on February 15, 1929, his left knee locked while he was getting into his automobile. The knee swelled and there was much pain, especially on its outer side. He was on crutches for two weeks, after which time the swelling and pain subsided. On March 14, he had a second "attack" followed by similar symptoms. I saw him four days later.

On examination I found the following: Left knee, absolutely normal in every respect. Motions normal. There is a little tenderness on the outer side of the knee and above the level of the patella. This tenderness is very constant. It embraces an area of about a twenty-five cent piece and apparent tumefaction can be felt in this region, although this is indefinite. No tenderness about the joint fissures. No effusion.

Numerous roentgenograms were taken and the following are the notes I wrote on our record: "X-rays taken in this office show a peculiar shadow which is present on all *x*-rays. (Several confirmatory pictures were taken.) It looks as though a small piece of wire were in the knee joint on its outer side and above the level of the patella. This shadow may represent the calcareous rim or edge of a cyst-like structure."

An exploratory arthrotomy was advised.

On March 21, 1929, the following operation was done: "Incision was made on the outer side of the knee joint above the patella. It was four and a half inches long. The knee joint was entered and after considerable search a small piece of very thin wire (corresponding to the *x*-rays) about five-eighths of an inch long was removed. The synovial membrane was sutured with plain catgut, the capsule with chromic catgut, and the skin with dermal. Posterior splint applied."

The piece of wire was embedded immediately below the synovial membrane. It could be barely seen glistening through this membrane, and it was definitely recognized after it was examined in situ by the examining finger.

It was only a few days following the operation that the patient recalled that a few months previous to the onset of symptoms he was polishing a piece of metal. He used an electrically driven circular wire brush and said that while doing this he had the sudden sensation in the knee as though he had been stung by a bee. He noted no blood on his clothes, forgot about it, and in no manner associated the onset of his knee trouble with this incident.

The following conclusions may be drawn:

(a) Any abnormality, as seen on a roentgenogram, however slight it may be, should attract attention. It should be followed by numerous confirmatory pictures until a definite conclusion has been arrived at.

(b) A foreign body, usually metallic in nature, can enter a joint without the patient being aware of it. This is especially the case with needles.

(c) Mr. Lane's injunction to use only instrumental technic in joint surgery is a good one and is followed by us. But there are occasional exceptions. If I had not put my gloved finger into this man's knee joint, I would not have found the offending foreign body.

DISCUSSION

DR. H. B. SWEETSER (Minneapolis): I just want to mention the difficulty of finding these foreign bodies and think Dr. Geist is to be congratulated on finding this so readily. To search for a needle or small metallic body in tissues is one of the most exasperating things. I have taken out small needles in the dark room under the fluoroscope.

DR. GEIST: There is not much to add, except that in obscure joint lesions we will occasionally find a foreign body at the bottom of the trouble.

D. EVERETT K. GEER (St. Paul) read his thesis entitled "Evulsion of the Phrenic Nerve for Pulmonary Tuberculosis." After the reading of the thesis Dr. Geer showed several lantern slides. (Thesis to appear later in MINNESOTA MEDICINE.)

DR. MCCLOUD (St. Paul): I am sure we all enjoyed very much hearing this excellent thesis of Dr. Geer, and I am going to ask some of those associated with him in the work to discuss it first.

DR. L. E. DAUGHERTY (St. Paul): Practically all of the phrenicotomies we have done have been preliminary to or following thoracoplasty. We have performed the operation of thoracoplasty on 115 cases. The last 50 have had phrenicotomies. It has seemed to me that the results obtained in the last 50 cases have been better and their symptoms have subsided more promptly than those on whom a phrenicotomy was not done. Considering the large number of anatomical variations in the course of the phrenic nerve, we have been very fortunate. In only two cases were we unable to find

the phrenic nerve. One of these cases had had a previous thoracoplasty and I can easily understand how the anatomy of the phrenic nerve may be disturbed by thoracoplasty. Dr. Sauerbruck has reported a case of fatal hemorrhage following a phrenicotomy. It is probable that the phrenic nerve was so intimately associated with the subclavian vein that when the nerve was evulsed the vein was torn. When we first started this work Dr. Gardiner suggested the use of the battery in order to identify the phrenic nerve. By stimulating the phrenic with the battery and by placing one hand on the upper abdomen, the nerve is easily identified by the sudden contraction of the diaphragm which can be easily felt. I have been particularly impressed not only by the anatomical variation in the location of the phrenic nerve but also by the size of the nerve. In some cases it appears to be the size of the lead of a pencil and in others it is much smaller. Dr. Gardiner, my associate in this work, has performed the operation of phrenicotomy in most of the cases which I report and I would like to hear from him.

Dr. E. M. Jones (St. Paul): I had some experience with a few of the earlier cases of Dr. Geer's series. I think that technically phrenicotomy is not a difficult procedure. The most important thing is the identification of the nerve, and this can be done by using a current to stimulate the nerve or by placing the patient under a fluoroscope and stimulating the nerve by grasping it with forceps. Another very important technical procedure is the slow evulsion of the nerve. After the nerve has been cut, the distal end should be grasped by forceps and with steady traction the nerve is very slowly rolled around the instrument. If there is a great deal of resistance and too much force is used some irreparable damage may be done.

Dr. D. G. Gardiner (St. Paul) (by invitation): I am indebted to Dr. Geer and Dr. Daugherty for seeing and operating these cases, and to this Society for the opportunity of discussing this paper. I think in seeing these slides of Dr. Geer, they more or less speak for themselves. When we tackle the subject of tuberculosis from the surgical standpoint, I think that any procedure that has to do with the collapse of the lung must be considered seriously. In these cases we have three sides of the chest to deal with, i.e., the thoracic, the diaphragmatic and the mediastinal. Any procedure which tackles the diaphragm itself seems to me as efficacious as any procedure which tackles the thoracic cage itself. Dr. Geer spoke about adhesions, which we used to think were contraindications to phrenicotomy. We have done a number of these at Ancker Hospital recently and in some we have found the diaphragm becomes fixed and doesn't move, and that accounts for lung relaxation obtained even without securing an actual rise of the diaphragm.

One point Dr. Geer mentioned in one case of far advanced tuberculosis that died. I was fortunate enough to see the post-mortem of this patient. There were very dense intra-abdominal adhesions which were adherent to the diaphragm on the peritoneal side. So we might well look to the peritoneal condition when the diaphragm does not rise. Dr. Geer also mentioned the fact that the twelfth intercostal innervates the diaphragm. We don't know but that the intercostals may take on a compensatory function. I feel that may be a factor. We have had cases in which we felt we had gotten the entire phrenic nerve without question and sometimes we have seen a late return of function.

Dr. Geer mentioned the fact that Mattson has dissected 112 cadavers. I did not feel very good about that when he told me the day before I took out a nerve that Mattson had found one case in which the nerve transfixed the subclavian vein and two others in which the nerve wound itself around the vein before going into the chest. However, we have not found anything of that sort yet. We have had no fatalities in our cases.

Dr. Jones mentioned the fact that this work is sometimes done under the fluoroscope. Dr. W. Meyer of New York has had to re-operate several cases in attempts to evulse the phrenic nerve. After identifying what he thought was the phrenic nerve he injected it with alcohol and did a fluoroscopy and found almost an immediate fixation of the diaphragm; he then proceeded to evulse the nerve. Undoubtedly fluoroscopy does help in unusual cases.

Dr. Daugherty spoke of finding the phrenic nerve after thoracoplasty. I have operated only one case after thoracoplasty and was agreeably surprised to find that it was the easiest case. Most men have the experience of hunting for the nerve and not finding it. Dr. Daugherty also spoke of some of the anomalies that have occurred and I myself have had one case where the nerve was buried in the substance of the muscle. Recently we found a number of cases in which the transverse colic artery and accompanying veins, lying high, have hidden the nerve and just this morning we had a case in which the phrenic nerve was lying behind this vessel.

In regard to post-operative treatment, there is practically none necessary. We have had no deaths, but we have had two cases which were rather unusual, that of subcutaneous emphysema. In one case (the man has subsequently died of his tuberculosis) we noticed that the next day after the operation he had a rather wide area extending up to the angle of the jaw, down over the chest and into the posterior cervical region. In another case it was much more limited. We were rather puzzled as to how this might occur. Perhaps the mediastinal pleura was torn. However, both of these cleared up. We sometimes see this condition after chest trauma. These two have been the only complicated cases that we have had.

In one case I thought I found the phrenic nerve; I got a pseudo reaction, and evulsed the nerve. The next day we found she had a Horner's syndrome. This evidently was only partial because it has cleared up to some extent since. In one case which I missed, and in which Dr. Daugherty missed, we both felt the nerve was absent entirely, as does occur, or else it was not lying in the normal position.

Dr. H. L. Taylor (St. Paul): These surgical efforts to help us in our care of the consumptive simply show how we are glorifying rest in the treatment of tuber-

culosis. That, of course, is the thing the surgeons showed us long ago in regard to joint and surgical tuberculosis but it is just as true of tuberculosis in the lung. The lung is constantly in motion. It is not confined or attached except at the hilum and it is shaking like a glassful of jelly all the time, the air coming in and going out with the movements of the body. But these surgical inventions have made it possible to collapse the lung or "splint" it and arrest its motion.

In the use of rest even outside of these cases where we do not use surgical intervention, our procedure has changed very considerably. Dr. Gerald Webb, who was recently here from Colorado, asked me if I had changed my procedure much in regard to the rest to which I subjected these patients. I remember when I first opened Pokegama I had a very strict rule that if a patient had a temperature of 100° that patient had to stay in bed for 24 hours after the temperature fell to normal. Now no one enters Pokegama unless he is put to bed for 6 weeks, whether he has fever or not, and he is fortunate if he gets up even when the 6 weeks have passed.

Dr. S. Marx White (Minneapolis): I realize the extraordinary interest which this subject has for the surgeon, but its interest is as great for those who are in charge of the prolonged treatment usually necessary in tuberculosis. Dr. Geer has given us a full, succinct account and valuable addition to our knowledge of this particular provision of rest in the treatment of tuberculosis. It might be interesting for the moment to think of some of the things that have been added to the treatment of tuberculosis by the surgeon. In the first place it was learned how great an aid it was to collapse the lung by means of pneumothorax. Jacobæus next found that, using an endoscope passed through an artificial opening in the chest wall, adhesions could be severed under visual control, and the use of pneumothorax was extended. Later bilateral pneumothorax was shown to be possible. Then with thoracoplasty (either partial or complete), with phrenicotomy, and also with multiple intercostal neurectomy, most of the movements of the hemithorax are brought under control by the surgeon. The physician who is treating the case has thus an adjunct which I think is an important one. Still the patient provides a medical problem under the prolonged care of the physician.

Dr. Geer (in closing): I have nothing to add except to thank the members who have discussed my paper.

Dr. Carl B. Drake (St. Paul) reported the following case of Massive Collapse of the Lung.

This case of massive collapse of the lung seems worth reporting inasmuch as the collapse occurred while the patient was confined to bed in the hospital and was not preceded by injury nor operation.

Mrs. F. E. F., aged 40, was admitted to the Ancker Hospital March 14, 1919, on the gynecological service, complaining of indefinite lower abdominal pain, weakness, and some fever. The pelvis was found to be normal and she was transferred to the medical service.

Aside from a pleurisy at the age of 21, and an appendectomy and cholecystectomy at 38, her history was unimportant. There was no history of exposure to tuberculosis. Her father and mother had lived to be over seventy and her husband and three children are living and well.

Upon examination the patient was found to be undernourished and running a daily fever of one to two degrees and a pulse of 90 to 100. The blood pressure was 112/50. The heart and lungs were normal on physical examination. The urine was normal. During her stay in the hospital the blood on two occasions showed a slight reduction in red cells and 23,000 and 22,000 white cells, with 47 and 36 per cent eosinophiles.

Two weeks after admission the patient complained of severe pain in the heart region aggravated by deep breathing. Two days later, examination showed the following:

The patient was lying comfortably in bed and did not appear to be dyspneic. The heart apex was palpable 8 cm. to the left in the fourth intercostal space and a cardiac impulse was visible and palpable 7.5 cm. to the left in the second interspace. The right heart border was at the midsternal line. Aside from a slight systolic murmur over the pulmonary area, the heart was normal. The left lung area was markedly impaired in resonance except at the base posteriorly, where the resonance seemed normal, while there was almost a flatness to percussion below the left clavivle. In the subclavicular region the vocal fremitus was increased, vocal transmission both to whisper and spoken voice was intensified, the expiratory breath sound was increased and many crepitant râles were heard. No râles were heard at the right lung apex. The spleen was palpable.

The chest *x*-ray showed a marked homogeneous opacity of the entire left lung and the heart definitely pulled to the left. There was also a slight mottling of the right apex and a faint line indicating interlobar pleural thickening in the right lung field.

A diagnosis of massive collapse of the lung was made, but the maneuver of turning the patient onto the right side and having her cough failed to expand the left lung.

Bronchoscopy was performed by Dr. Gardiner on April 12th. No mucous plug was found but the bronchi on the left side appeared somewhat narrower than normal. The *x*-ray taken just before bronchoscopy showed the left side less opaque and an *x*-ray taken ten days later showed no change in the left side and definite patches of infiltration in the right lung area. An *x*-ray taken April 30th showed a marked clearing of the left side, less infiltration on the right side and the heart only slightly drawn to the left. The last *x*-ray, taken May 20th, shows further clearing of both lung areas, a slight mottling of both apices, a slight generalized increase in lung design and the heart nearly central in position.

On July 8th the patient reported to the office. She had no symptoms, had gained 9 pounds since discharge from the hospital on June 1st, and physical and fluoroscopic examination failed to reveal any abnormal lung signs.

The differential diagnosis in this case is rather interesting. It was first thought that we were dealing with a case of pulmonary tuberculosis. The history of pleurisy, the fever of a degree or two which persisted for two months, the mottling of both apices, the crepitant râles, and the poor state of nutrition all suggested a tuberculous infection. Six sputum examinations, however, proved negative for tubercle bacilli, and recovery occurred. I think it likely that the patient had had an apical tuberculosis at one time.

The probability of some unusual lung infection such as actinomycosis was considered. Sputum examination, however, failed to show any such organism.

The picture was certainly not one of a lobar pneumonia nor of a pleurisy with effusion. Mediastinal displacement too was against either condition.

The case is one of massive collapse of the entire left lung. Whether an infection and enlargement of the left hilus glands was also present, possibly secondary to some lung infection such as a bronchopneumonia which was not recognized before the lung collapsed, is a question. One of the later *x*-ray films showed a definite scattered infiltration of the right lung indicating a definite lung infection which also may have been present in the left lung. This, however, followed the bronchoscopy. I cannot see how the patient could have recovered had hilus gland enlargement due to tuberculosis been the causative factor.

My opinion is that we were dealing with a case of massive collapse of the lung due to simple inanition and accompanying infection the cause of which was undetermined.

DISCUSSION

Dr. A. T. Mann (Minneapolis): This is very interesting to me, this massive atelectasis following a medical condition, for we are more familiar with it, though it is not so very common, in surgical conditions. Pasteur, about 1890, is the first one who called attention to these conditions and reported thirty-four cases following diphtheria. Some years later he also reported some cases after operation.

In regard to the mechanism, it is still in doubt of course, though there are some interesting speculations about it. In the first place it has been found that the bronchioles and even the air cells themselves are contractile. The bronchi and bronchioles have considerable striated muscle tissue in them. The lungs are an outgrowth of the digestive tract and apparently have retained some of the action of the stomach and bowels. Henderson and his co-workers have mentioned that there is at least a rhythmic contraction of the bronchi, bronchioles and air cells about once in 7 or 8 minutes which would help to empty them. Their innervation is by the vagus. The vagus is tonic in its action and I wonder whether that explains in part the reason why we have these things. A reflex through the vagus could very well help to contract the lung cells and bronchioles; then the remaining air will be very rapidly absorbed, taken out through the vessels in the cell walls.

As to the surgical cases, most of them have occurred after ether, or prolonged operations in which the respiration is reduced considerably. The reduction of respiration and the stimulation through the vagus, together, could bring about the condition of atelectasis. We saw in these *x*-ray pictures the condition itself; it looks like a solid body. There is no air in the pleural cavity and practically no air in the air-cells themselves. The chest, diaphragm and mediastinum are all sucked in toward the collapsed lung.

As to the cause being a plugging of the bronchus with mucus, that doesn't seem to me to hold water very well. In the bronchoscopic trials for clearing the bronchus, they only bring out a little mucus such as would be there after the bronchioles had closed down; then there would be a plugging so that the lung itself, after the air had gone out of it, would not fill.

And that brings us to the treatment. Methods have been suggested here. Putting the patients on the well side and having them cough has succeeded in a great many of the surgical cases. The first case in which that happened was a man in St. Louis and this case had resulted after injury about the hip. When fluoroscoping him, the man was in so much pain that they turned him on the other side; the man had a sudden severe pain, coughed, and the lung opened out while they were looking with the fluoroscope. The interest for us surgeons is to give them change in position after an operation, in an attempt to avoid lung complications. The next thing, to my mind, is to give them carbon dioxide inhalations. The respiratory excursion is much reduced after prolonged ether and Nature's method of increasing respiration is the carbon dioxide that is manufactured within us. It has been found that administration of CO_2 will greatly increase the excursions of the lungs. Again, Henderson found that the administration of CO_2 gas was the treatment for carbon monoxide poisoning, so he and his co-workers developed the CO_2 inhalations in carbon monoxide poisoning and it is still the best treatment known. It increases the excursions, it increases the volume of inspired and expired air 30 or 40 liters per minute. And that opens up the lungs, and it has prevented almost all the cases of pneumonia following carbon monoxide poisoning, of which there used to be a large number. We can avoid a great many of our pneumonias probably if we give CO_2 after ether administration. Of course some rare cases have occurred after a local anesthetic and in these the pneumogastric nerve reflex is the probable cause, but the carbon dioxide inhalations will expand the lungs and give them better excursion even in these cases. In a large series of post-operative cases studied by Scott, some 2,000 cases in which carbon dioxide had been administered following the anesthetic and some 2,000 cases in which it had not been used, it was found that the occurrence of massive atelectasis of the lungs as well as post-operative pneumonia was considerably less in the series in which carbon dioxide had been given. My suggestion is that in our hospitals give carbon dioxide inhalations for twenty or thirty minutes after our ether administrations.

Dr. Gardiner (by invitation): I saw this case at Dr. Drake's suggestion. We were impressed with the fact that we were dealing with a massive atelectasis.

I could not help but recall a case which I had seen with Dr. Wm. Lerche, with whom I was formerly associated. This occurred in a child two years of age. This child had a rather acute febrile attack and suddenly became quite dyspneic. The mother thought the child had aspirated a foreign body. *X*-ray showed complete atelectasis of the upper lobe on one side. Dr. Lerche was asked to see the child and bronchoscoped it and could see only a flat bronchus, so flattened that in looking into it you could see the posterior wall flattened out against the anterior. Then he realized that he was dealing with some extra-bronchial pressure. I think he subsequently published that case report. The child went through a course just as this one of Dr. Drake's. Dr. Finklestein, of Berlin, was here at the time and saw the child in consultation and agreed that the child had an acute tuberculosis with acute lymphadenitis. The child was given rest and an anti-tuberculosis regime and has been perfectly well since.

When I saw the bronchus in this case I was reminded of Dr. Lerche's case, for while they did not meet here, they were very close together.

As to what Dr. Mann said in regard to massive collapse and the mucous plug, I feel that there is a good deal to that. Dr. Jackson has found a tenacious mucous plug time and again. He has gone so far as to have some of his workers aspirate some of these patients, subject animals to operation, and inject the material intrabronchially into these animals, and in some of the animals massive collapse resulted.

I still feel that Dr. Drake's case is one of overwhelming infection—whether tuberculous or not, I do not know—with an acute tracheobronchial or bronchopulmonary lymphadenitis. Her subsequent recovery would seem to fit in with such a picture.

Dr. H. B. Zimmermann (St. Paul) reported a case of Spinal Cord Tumor, with operation, referred by Dr. Hengstler of St. Paul.

DISCUSSION

Dr. A. Schwyzer (St. Paul): This is a very pretty case. I had the good luck to see the result a few days ago. The woman walked remarkably well. The idea of injecting lipiodol below and tilting the patient seems a good procedure. The operating of these cases requires the utmost delicacy not to injure the cord the least bit. When you have a complete obstruction of the spinal canal even a difference of 1 mm. is liable to cause much damage to the cord. The case being a meningioma I would say it probably was of rather solid consistency and could be freed very readily from the surroundings. Is that not true?

Dr. Zimmerman: It was quite solid and came out whole.

Dr. Schwyzer: Fortunately, when we have to deal with a meningioma it is well outlined. A week ago I had a case of a large meningioma below the first temporal gyrus and it peeled out readily. If we have a spinal cord tumor within the cord, the condition is very, very much more dangerous; and then a good maneuver (because the least little damage is very serious) is to just incise the cord down to the tumor and do nothing further for a time but close the dura with very finest silk and wait two or three weeks. The cord will gradually deliver the mass more to the surface and it can be removed at a second operation. We all hate to operate a second time, but in a case of this kind what we gain by it is worth taking the drawbacks into the bargain.

Dr. A. Schwyzer (St. Paul) reported the following case, and autopsy:

I made an autopsy on a woman this noon. This woman was brought into the hospital after having been in misery for nine days. She had not seen a doctor until that afternoon. She was 71 years old. She had been vomiting almost constantly, and appeared very bad on admission. The vomitus looked and smelled feculent. Washing out the stomach did not yield much material but very soon after the lavage she vomited feculent material again. She was in no shape for any prolonged examination and it seemed the only thing the old lady could stand would be an enterostomy and we would then have to see if this gave relief. With local anesthesia we made an enterostomy with a large-sized catheter through a McBurney incision. The lowest ileum was very ballooned. We got considerable gas out through the tube and some fluid, but had to coax it with small injections of water. There was no sign of peritonitis. We figured that we had some obstruction and a partly paralytic condition of the small intestine from overdistention. She was given pituitrin, atropin, and later on was given intravenously strong salt solution. (In two cases on the verge of going out, we had given 50 c.c. of 30 per cent salt solution intravenously and it started peristalsis.) The patient grew markedly worse though we always had had some drainage and escape of gas from the tube. Day and night the nurse injected 30 c.c. of 5 per cent salt solution every half hour into the enterostomy tube. Two days after the enterostomy we decided to make a second attempt. Under spinal anesthesia we opened the abdomen large enough to admit the hand. The stomach was empty. She had vomited whatever came back from the duodenum; the jejunum and ileum were very much distended. We did not dare to do much. Another enterostomy was made, a little higher up, and again with a catheter; and again we had escape of gas. We then milked out the contents from many loops. Both the new and the old stoma worked. Still she did not do any better and she died this morning, three days after this second operation. I did not give eserin, as the patient seemed too weak at any time to stand its depressant effect.

The autopsy showed that these two enterostomies had not worked sufficiently. While they were patent and would allow of drainage under some pressure, the guts in the immediate neighborhood of the tubes were ballooned. It occurred to me that the opening was not quite wide enough and if I had the case to do over, with this knowledge, I think I would put in a considerably larger tube. The other findings were an enormous cecum, a ballooned descending colon, and in

the pelvis a big mass. (Diagram on blackboard.) Here was a large mass which looked like a carcinoma, but it was not carcinoma. We had a thick-walled abscess in the wall of the rectosigmoid. The gut was completely strictured at this level. I could not find the opening with a probe. After we had cut out the whole area we found at the same level as the thick-walled abscess a pocket with a fecolith in it of about half by one-third inch diameter. This diverticulum also had thickened walls.

It was not possible to make an *x*-ray when the patient came in in the night; she was in no shape for delay; nor would it have done much good. With the very ballooned small intestine it did not seem sufficient to make a colostomy. I think if I had to do it over again I would put in a very large tube or take the gut to the surface and cut it open.

DISCUSSION

DR. H. B. ZIMMERMANN (St. Paul). I had a case day before yesterday with a baby with acute obstruction and had the same experience. In opening the intestine we emptied one loop of gas and the other remaining loops did not drain. The main thing is to get air out of the small intestine. This baby was vomiting intestinal contents. I injected salt solution, made a laparotomy, and the bowels pushed right out. I aspirated gas and liquid from that loop of the bowel and continued that until I had made twelve openings in the bowel. By that time the abdomen was pretty well collapsed. That was day before yesterday, and today the baby is having normal bowel movements. I think the important thing to do is to collapse the intestine, and think there is much less trauma in making repeated openings. This is heroic treatment but sometimes heroic treatment is necessary in these cases.

DR. A. E. BENJAMIN (Minneapolis): I have a case at the present time that had obstruction just above the sigmoid with diverticulitis and abscess about as Dr. Schwyzer described it. What I did in the case was to bring the descending colon up through the abdominal wall. I then dissected a fascial strip from the external oblique, passed it through the mesentery and stitched it to the opposite side of the wound, using it as a bridge to hold the colon up. I then stitched the peritoneum around the colon. Through an opening in the colon I passed a tube and washed the bowel out thoroughly. I stitched the tube in place. A gastric lavage was then done every two to four hours until the alimentary canal was thoroughly cleansed and there was no more regurgitation, after which the bowel then acted through the colostomy opening.

The patient gradually recovered, but later developed an abscess on the right side which had dissected its way from the area of the abscess due to the diverticulitis on the left. The patient still has the colostomy opening. She is up and around a good part of the time, but complains of some pain occasionally.

These cases are very interesting and always a source of annoyance to the surgeon and attending physician.

DR. SCHWYZER (in closing): I think what Dr. Zimmermann said is excellent. We have to get rid of the contents of the intestine. Of course, this patient was an old lady very far gone. We did milk out the intestines. The autopsy showed that the guts were not agglutinated from our manipulations; they were free. The fact that the guts have to be emptied thoroughly, and more thoroughly than we dared do in our case, is a very important one. Whether it is easier or better, particularly in an old patient, to tap many loops or to milk them down through a tube, is a question.

The meeting adjourned.

CARL B. DRAKE, M.D.,
Secretary.

PROGRESS

Abstracts to be submitted to Section Supervisors.

Members are urged to abstract valuable articles which they run across in their reading and send the abstracts to the physicians in charge of the respective sections. In order to avoid duplication it would be well to communicate with one of the section supervisors before the article is abstracted.

SURGERY

SUPERVISORS:
DONALD K. BACON,
LOWRY BLDG., ST. PAUL
VERNE C. HUNT,
MAYO CLINIC, ROCHESTER

THE OPERATION FOR UNDESCENDED TESTICLE: Arthur Dean Bevan (Ann. of Surg., Vol. XC, No. 5, 1929, pp. 847-863). Doctor Bevan emphasizes early operation in these cases; stating any time after the first or second year as preferable. The four reasons for this view are:

1. The structures are more pliable and yielding.
2. The cord is more easily lengthened so the organ can be brought down without tension.
3. There is less danger of doing enough injury to cut off the blood supply.
4. There is a better chance for normal development.

The latter opinion is based on the work of Doctor Carl Moore of the University of Chicago, who found that the testicles of young healthy animals when placed in the peritoneal cavity gradually degenerated and never developed to full function, this being due to the difference in temperature. On the other hand, when these same testicles were retransplanted to their normal position in the scrotum they recovered their full development and function in a short period of time.

Important points in the surgical procedure are:

1. The use of delicate instruments and catgut.
2. The use of normal saline and hypodermic syringe in separating the peritoneal vaginal process from the cord.

3. Ligation of the upper vaginal process at the internal ring.

4. The use of the lower peritoneal covering as a tunica vaginalis.

5. Purse string suture at the neck of the scrotum to maintain the testicle.

6. The canal is closed with the cord deeply situated; the transversalis and internal oblique are sewed to the shelf of Poupart's ligament over the cord.

C. W. Mayo, Jr., M.D.

PEDIATRICS

SUPERVISORS:
CHESTER A. STEWART,
LA SALLE BLDG., MINNEAPOLIS
ROY N. ANDREWS,
MANKATO CLINIC, MANKATO

FORTY-TWO CASES OF APPENDICITIS IN CHILDREN OCCURRING DURING AN EPIDEMIC OF UPPER RESPIRATORY TRACT INFECTION: Harold J. Freedman, M.D., Boston (Arch. of Ped., October, 1929, Vol. XLVI, No. 10). Concerning the age incidence, the youngest of a group of forty-two were two children of two years, both of whom had a ruptured appendix. Abt reviewed eighty such cases, in which twenty patients were less than three months of age.

Although in a series the temperature is moderately increased in acute non-perforated appendicitis and definitely increased in those cases showing perforation, we must not overlook the important fact that in individual cases the temperature is of little decisive value. A table shows the average pulse to be elevated but very high in the cases with perforation. Nevertheless, the pulse is a more accurate sign of toxicity than the temperature or the leukocyte count.

LEUKOCYTE COUNT

George Aldous laid great stress on the white blood count and said that all doubtful cases with a count of 15,000 to 30,000 should be explored. Russell Fowler reported an average count of 19,100, with an average polymorphonuclear count of 89.7 per cent, in a series of cases. Alfred Noehren believed the differential leukocyte count to be of more value than the count itself. A leukopenia in the presence of a pathological appendix is indicative of marked toxicity.

As a rule, closely following the first attacks of pain, there are gastrointestinal symptoms. The high percentage of those who vomited is striking proof of the extreme importance of this symptom. We must remember the possibility that many acute appendices occur as a consequence of upper respiratory infection. Brenneman emphasized the value of localized physical findings such as tenderness, spasm, or rigidity, and remarked that the white blood count was not a great contributory finding so far as differential diagnosis was concerned.

1. Perforation was frequent in the earlier years.

2. The finding of an increase in the white blood cells in the urine is quite common in appendicitis.

3. Post-operative discomfort was reduced in the toxic cases by repeated administration of saline hypodermoclysis.

4. The finding of localized tenderness and spasm in the abdomen is more important than any other clinical signs in the diagnosis of appendicitis.

R. N. Andrews, M.D.

INFANTILE PYLORIC OBSTRUCTION—PRELIMINARY REPORT OF ITS ALLERGIC NATURE: Milton B. Cohen, M.D., and Joseph Breitbart, M.D., Cleveland (Amer. Jour. of Dis. of Children. October, 1929, Volume 38, Number 4). In spite of much clinical observation, infantile pylorospasm and pyloric stenosis remain obscure in etiology and in their relationship to each other.

If, on the administration of atropine and on a thick diet, the child improves, the case is one of pylorospasm; if no improvement follows these measures or if the symptoms continue to grow worse despite them, a diagnosis of organic obstruction is made and operation is advised. Pathologic evidence is borne out by the clinical fact that most of these infants take food normally and show normal growth curves for from a few days to a few weeks, when suddenly the symptoms begin.

It can be seen that the vomiting and visible peristalsis common to this disease result from pyloric obstruction, either completely spasmodic or combined organic and spasmodic in origin, with the symptoms following completion of the obstruction by the smooth muscle spasm with its accompanying edema and exudation.

Ratner has demonstrated experimentally that allergic antibodies can pass through the placenta of the guinea pig and passively sensitize the offspring. He has also succeeded in administering the sensitizing dose of horse serum to the guinea-pig fetus by injecting the substance into the mother a few days before parturition, and has demonstrated that sensitization, which was not present at birth, developed within a period of one month.

The only essential difference between those cases of pyloric obstruction which show hypertrophy and those which do not show it may be in the time of onset of sensitization and its duration. Patients with organic pyloric obstruction are probably sensitized in utero early in prenatal life. Repeated shocks probably occur during prenatal life with the resulting secondary hypertrophy. After birth the child may develop normally for a few days until it receives a dose of the specific allergen, and this institutes further spasms and edema with its resultant signs.

(1) The pathologic condition in infantile pyloric obstruction is probably identical with that in allergy.

(2) Evidences of allergy are present in the majority of cases of infantile pyloric obstruction.

(3) Depending on the period in life at which sensitization occurs and the frequency and severity of the shock reactions, there may be pylorospasm with or without organic obstruction.

R. N. Andrews, M.D.

ROENTGENOLOGY

SUPERVISORS

LEO G. RIGLER

MINNEAPOLIS GENERAL HOSPITAL, MINNEAPOLIS

A. U. DESJARDINS

MAYO CLINIC, ROCHESTER

X-RAYS IN THE DIAGNOSIS OF EARLY CARCINOMA: H. Haudek (Brit. Jour. of Radiology, V. 2, p. 421, September, 1929). Cancer of the stomach is still a very fatal disease due chiefly to late diagnosis. There are four principal causes for this:

1. The latent period during which the patient has no symptoms or they are not localizing.
2. Delayed *x*-ray examination due to reluctance of both doctors and public to utilize the *x*-ray method.
3. Faulty *x*-ray investigation, that is, insufficient, careless examination by untrained individuals.
4. Faulty methods in the radiological investigation.

The author discusses chiefly the latter point and makes suggestions based upon his experience. The use of the barium-filled capsule to determine the slightest hindrance in the lower end of the esophagus or cardia of the stomach is strongly advised.

The use of a small amount of a thick barium emulsion for the examination of the contracted stomach and its folds is advocated. In this way very small ulcer niches, tumor craters and small filling defects may be found. Films are also made with pressure applied by means of pads. The completely filled stomach is then observed in the usual way. The prone position must always be used.

The author has been able by the *x*-ray method to determine the presence of slight carcinomatous changes in the gastric wall—very early diagnoses in a few cases. Numerous cases including benign tumors of the stomach are cited to support this assertion. In the postwar period he has not failed to diagnose carcinoma when it was present in any case. The possibility that carcinoma might be erroneously diagnosed when no lesion existed should be considered. He has had no failures in this respect. The distinction between a prepyloric benign ulcer and a malignancy, however, is much more difficult, at times impossible. The frequency with which ulcers in this region undergo malignant degeneration should make their treatment surgical so that the distinction is not so important.

LEO G. RIGLER, M.D.

PULMONARY TUBERCULOSIS OF INFANTS AND CHILDREN: Rolla G. Karshner, M.D. (Amer. Jour. Roent. & Radium Therapy, Sept., 1929, Vol. XXII, 226-234). The author presents a study of proved pulmonary tuberculosis at the children's hospital, most of the cases having been studied for weeks, months or years prior to postmortem examination. The classification used is based on the appearance and distribution of the infiltrations on the roentgenogram as primary glandular, hilus, miliary and adult tuberculosis.

The focal primary lesion of tuberculosis is almost always undetected. It is seen later as a calcified remnant, usually in a lower lobe. Tuberculosis of the intrathoracic lymph nodes is generally diagnosed roentgenologically. The enlargement is often extreme in infants. Tuberculous hilus glands may cause all the phenomena commonly associated with foreign body in the air passages. In children, it can rarely be stated from the roentgenogram whether disease of the intrathoracic nodes is the cause of the symptoms present. Calcification rarely, if ever, occurs in other than tuberculous hilus glands. Hilus tuberculosis originates at the root of the lungs and extends outwards a variable distance. Miliary tuberculosis may be a terminal phenomenon in any type of tuberculosis. Occasionally healing occurs. In infants it usually accompanies tuberculous meningitis. The adult form is rare before the twelfth year.

JACOB SAGEL, M.D.

SIGNIFICANCE OF GAS UNDER THE RIGHT DOME OF THE DIAPHRAGM: E. P. Pendergrass, M.D., and Elizabeth Kirk, M.D. (Amer. Jour. Roent. & Radium Therapy, Sept., 1929, Vol. XXII, 238-246). The authors present several cases in which the finding of gas under the right dome of the diaphragm by *x*-ray examination helped to crystallize the opinion justifying subsequent operation. The demonstration of gas is made by films taken in the lateral and anteroposterior positions, especially in the lateral position. In the case of gas within a subphrenic abscess, the gas usually lies above a fluid level which has the shape of a segment of a circle and there is a definite wave effect of the fluid after shaking the patient. In the case of hepatoptosis, the haustrations of the colon can be seen under the dome of the right diaphragm. Transposition of viscera with the stomach on the right side can be demonstrated by a barium meal and a plate showing the heart on the same side. Gas under the dome of the diaphragm following an operation can be ruled out by the history of the preceding operative procedure.

The article is illustrated by many plates showing the conditions mentioned. An extensive bibliography is appended.

JACOB SAGEL, M.D.

AMERICAN PUBLIC HEALTH ASSOCIATION OFFERS LIFE MEMBERSHIP

The American Public Health Association has recently arranged a life membership status for its members and fellows. Applications must be approved by the committee on eligibility and accepted by the Governing Council. Thirty life members were created at the recent Minneapolis convention. The cost of life membership is $100.

EYE, EAR, NOSE AND THROAT

SUPERVISORS:
VIRGIL J. SCHWARTZ,
PHYS. & SURG. BLDG., MINNEAPOLIS
ARTHUR C. DEAN
CROOKSTON CLINIC, CROOKSTON

CONDITIONS OF THE OPTIC NERVE CAUSED BY DISEASE OF THE SINUSES: Thompson, New York (Arch. of Otolaryng., Sept., 1929, Vol. X, No. 3, p. 248). Of the various conditions of the eye due to disease of the sinuses, none are so well recognized or established as disease of the optic nerve. There are today few ophthalmologists who on seeing an inflammation of the optic nerve do not send the patient to a rhinologist for a report as to the presence of ethmoiditis or sphenoiditis. From these reports it is evident that there is a large class in which no intranasal symptoms occur. These are known as the latent sinus infections. In many of these cases results have been so good from operations that the practice of operating on these so-called latent sinuses in the presence of suggestive symptoms in the eye became established and continues until the present time. It became evident that the nasal symptoms could not be depended upon.

There are three types of optic nerve disease occurring as the result of sinus disease: (1) Retrobulbar neuritis; (2) plastic neuritis; (3) sudden functional depression with no change in the appearance of the optic nerve.

It seems that in the sinus infections with pus the mucosa is thickened and walls off absorption, while in the latent cases the mucosa is thin and the infection spreads directly into the bone. When the optic nerve is concerned the condition is almost always in the posterior ethmoids and sphenoid.

ARTHUR C. DEAN, M.D., F. A. C. S.

HEALTH WORK AT A STATE FAIR

The September issue of the *Nebraska Medical Journal* has the following editorial on health work at the State Fair:

"As you know, the state health work and exhibits at the State Fair are now under the supervision of the Nebraska State Medical Association. We have been putting in some extra work on the proposition this year, and are really desirous that the doctors of the state visit that department.

"We have the examinations for adults, the Four H Club, the babies, and some exhibitions, lectures, and reels on contagious diseases, particularly diphtheria prevention. In the babies' department we are planning for some reels and slides in addition to the examinations of the babies, and possibly some lectures. We have really put a good deal of work on the proposition this year and are desirous of constructive criticism."

BOOK REVIEWS

Books listed here become the property of the Ramsey and Hennepin County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

TULAREMIA. Walter M. Simpson, M.S., M.D., F.A.C.P., Director Diagnostic Laboratories, Miami Valley Hospital, Dayton, Ohio; formerly Senior Instructor in Pathology, University of Michigan. 162 pages. Illus. Cloth, $5.00. New York: Paul H. Hoeber, Inc., 1929.

CLINICAL MEDICINE FOR NURSES. Paul H. Ringer, A.B., M.D., formerly Chief of Medical Service of the Asheville Mission Hospital, Asheville, N. C. 330 pages. Illus. $3.00. 3rd revised edition. Philadelphia: F. A. Davis Company, 1929.

PRACTICAL MASSAGE AND CORRECTIVE EXERCISES. Hartvig Nissen, Late President, Posse Normal School of Gymnastics. 5th edition revised and enlarged by Harry Nissen, President, Posse Normal School of Gymnastics, Boston. 271 pages. Illus. $2.50. Philadelphia: F. A. Davis Company, 1929.

THE NUTRITION OF HEALTHY AND SICK INFANTS AND CHILDREN. E. Nobel, Professor of the University and First Assistant; C. Pirquet, Late Professor of the University and Director; and R. Wagner, Associate Professor and Second Assistant, University of Vienna, Children's Hospital. 2nd revised edition. Authorized translation by Benjamin M. Gasul, B.S., M.D., Consulting Pediatrist, Municipal Tuberculosis Sanitarium, Chicago. 243 pages. Illus. $3.50. Philadelphia: F. A. Davis Company, 1929.

DISORDERS OF THE SEXUAL FUNCTION IN THE MALE AND FEMALE. Max Huhner, M.D., Chief of Clinic, Genito-urinary Department, Mount Sinai Hospital Dispensary, New York City. 3rd edition. 342 pages. Philadelphia: F. A. Davis Company, 1929.

ANGINA PECTORIS. Harlow Brooks, M.D., Emeritus Professor of Clinical Medicine, New York University. (Harper's medical monographs.) 172 pages. Price, $2.50. New York and London, Harper & Brothers.

This book is not a review of the literature already published, but a thorough study of the subject from the consulting physician's viewpoint. Dr. Brooks assumes a new outlook for angina patients, or those predisposed to this disease by reason of heredity or other etiological factors, advancing the idea that angina may not be fatal, or if treated in the early stages the result may be regarded as an effective cure. It is possible, he contends, to so regulate the lives of those predisposed to this condition that they may escape it.

The author has discussed the etiology so far as it is known at the present time and has given careful consideration to the pathology and pathologic physiology as developed from his observation. Symptomatology, in-

cluding obscure types of syndrome, differential diagnosis, especially between true and false angina, and detailed treatment, are presented clearly and comprehensively.

A. E. CARDLE, M.D.

A SURGICAL DIAGNOSIS. J. Lewi Donhauser, A.B., M.D., F.A.C.S., Clinical Professor of Surgery, Albany Medical College. 799 pages. Price $10.00. New York: D. Appleton, 1929.

In the preface, the writer gives an excellent illustration of how surgical diagnosis should be taught to medical students.

This book was written essentially for medical students, hospital residents and general practitioners. It contains a vast amount of data and numerous charts. There are etiologic charts, regional and anatomic charts and differential charts which, if taken in conjunction with history taking and examinations, should be of vast assistance.

From the use of the large number of charts alone one would not have a great deal of trouble in making a surgical diagnosis.

O. S. WYATT, M.D.

OSTEOMYELITIS AND COMPOUND FRACTURES. H. Winnett Orr. 208 pages. $5.00. St. Louis; C. V. Mosby Co., 1929.

One is interested in reading this book by Dr. Orr, dealing with the treatment of osteomyelitis and compound fractures, to find that all the steps or parts of his method are ones previously advocated, in part at least, by various surgeons from the time of Paré. Dr. Orr is to be commended highly for the very effective way in which he has brought these ideas together and emphasized them.

The first chapter, dealing with the history of antisepsis from the time of Lister, adequately sets forward the ideal method of treating wound infections. This method briefly is to allow the wound as great an opportunity to heal as is possible, by not changing the dressings too often and by not using a wide variety of irritating antiseptics. It is in this frequent dressing of wounds that the damaging secondary infection is produced. Dr. Orr's reference to Dr. Alonzo Clark, "Don't treat your patients too much. Let them get well if they can," is very timely.

The second chapter deals with the condition in fracture and injection treatment prevalent during the war. The changes in the viewpoint of the war-time surgeon is immobilization of fractures, treatment of infections, and development of "orthopedic viewpoints" is most interesting.

Chapter three deals with osteomyelitis specifically. Numerous references to Dr. Starr's excellent work on osteomyelitis are made and full description of the treatment of osteomyelitis by Orr's method is given and numerous case reports are used to illustrate various types of infections or complications in the course of the disease. The results of treatment are most encouraging and instructive.

Chapter four, dealing with the application of Orr's method to the treatment of compound fractures, is new and individual. It must be said that not many surgeons could enclose a compound fracture in plaster-of-Paris without feeling very nervous about the ultimate outcome. Dr. Orr shows very convincingly that his method is easy and most applicable in this type of case. Again, case reports are used to prove all assertions made and the end-results seem to be uniformly good.

Chapter five is devoted to the clinical results obtained by various surgeons using Orr's method. All of these reports undoubtedly are picked cases, show excellent results and prove conclusively all the good points of the Orr method.

The book is well illustrated with pictures and case reports and is a very readable one.

GEO. A. WILLIAMSON, M.D.

YOUNG MEDICAL MAN would like to take over some doctor's practice temporarily or permanently beginning January 1. Apply Dr. S. E. Horwitz, Iowa Methodist Hospital, Des Moines, Iowa.

FOR SALE—Standard *x*-ray equipment, combination table, fluoroscope, developing tank, Alpine lamp. G. L. Merkert, 1527 E. Lake Street, Minneapolis.

WANTED—302 Lowry Building. Physician to share fully equipped, nicely furnished office. Reception and examining rooms including light and telephone. $30 monthly.

WANTED—Single doctor, recent graduate, to do Contract Practice. Good salary. Address D-49, care MINNESOTA MEDICINE.

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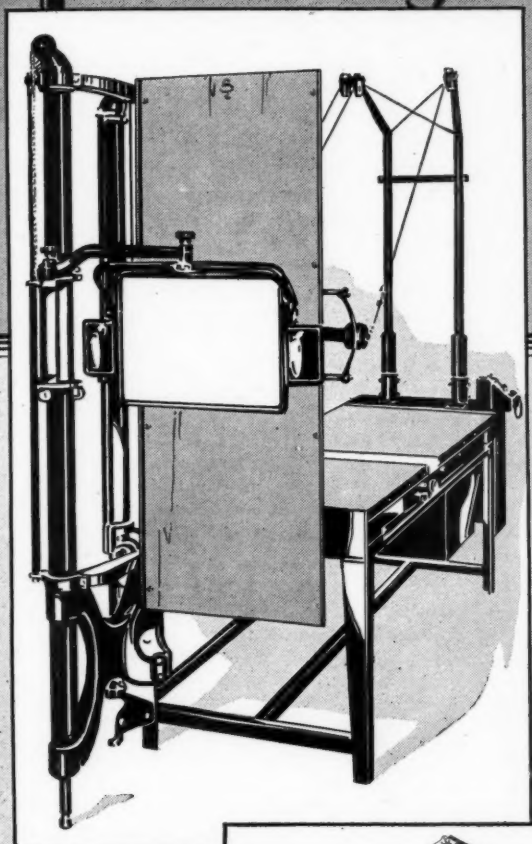

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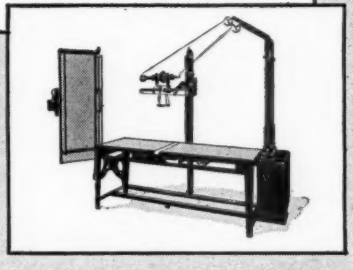
It consists of an oil-immersed transformer with an instrument panel of striking simplicity and accuracy, demanding no special training for its successful operation. The improved American Flat Potter-Bucky Diaphragm is mounted on a special track in the sturdy, beautifully finished X-Ray Table, to permit the making of radiographs of any part of the body without having to move the patient. The radiographic tube carriage can be quickly connected to the finely counter-balanced fluoroscopic screen, which has ample range for all forms of vertical fluoroscopy.



We shall be glad to mail you a booklet, telling what hundreds of successful physicians the country over have to say about the efficient TripleX Unit.

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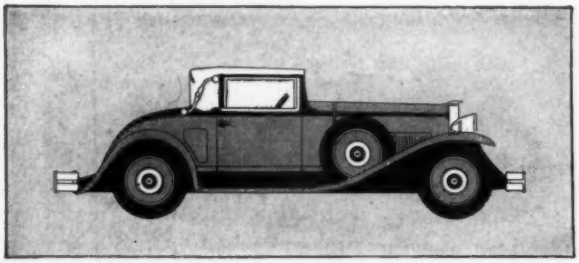

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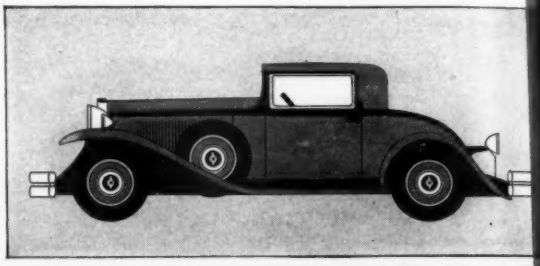

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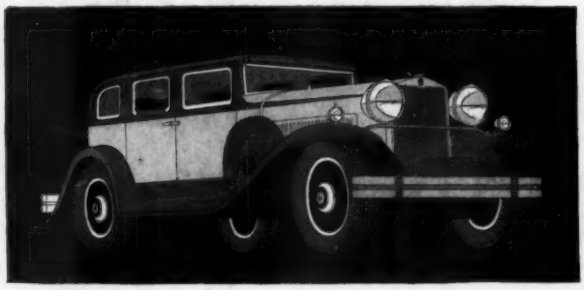

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